Health Risks from $PM_{2.5}$ and Ozone in Melbourne: Present and Projected Futures

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Declaration

I declared the original of this thesis. Otherwise indicated, all the work in this thesis is my own, conducted under supervision of Prof Robyn Lucas, Prof Keith Dear, Dr Martin Cope, A/Prof Steven Roberts and Prof Tord Kjellstrom through National Centre for Epidemiology and Population Health, the Australian National University.

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Date: 17 April 2014

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Abstract

In Australia, adverse health impacts of air pollution have still been reported despite relatively low pollution levels. There is a growing concern that climate change may have a detrimental effect on future air quality and impose additional air pollution-related health burdens. This thesis aimed to assess the impacts of future climate change on the health effects of air pollution in the Melbourne Region.

Firstly, the best approach was identified to estimate baseline relative risks for cardiovascular and respiratory mortality and emergency department (ED) visits associated with exposure to particulate matter with diameters of less than 2.5 micrometres (fine PM) and ozone. A Poisson regression model was fitted to estimate the baseline relative risks by using data during 1999–2008. A blending approach in which air pollution and weather data were generated by merging simulations and measurements was selected as it provided larger risk estimates, thought to indicate lower measurement error.

The blending approach was used further to explore whether there was evidence of a modifying effect of temperature on the air pollution-related health risks. The Poisson model previously developed was modified to include an interaction term for temperature strata. Non-uniform relative risks for fine PM- and ozone-related respiratory ED visits across the temperature range were observed, suggesting the effect modification of temperature.

Next, the baseline relative risks earlier estimated were used to predict changes in air pollution-related respiratory ED visits induced by climate change in the Port Phillip Region between 2065–2074 and 1996–2005. Two methods, with and without inclusion of the temperature modifying effect, were used. Without the temperature modifying effect, exposure to future ozone modified by the changing climate was estimated to cause an additional 60 to 110 respiratory ED visits over three summer months. An increase of 15 to 26 respiratory ED visits over the summer period was estimated due to exposure to fine PM. In winter, a reduction in fine PM-related respiratory ED visits was estimated based on three of four global circulation models. When using the method taking account of the temperature modifying effect, an increase in estimates of fine PM-related respiratory ED visits for summer and winter between the two periods was predicted, with a greater magnitude compared to the estimates predicted by the other method. A little difference in ozone-related respiratory

morbidity between the two decades was predicted for summer with inclusion of the temperature modifying effect.

Finally, the sensitivity of the estimated changes in respiratory morbidity to uncertainties in non-climate factors was explored. Among the factors examined, future population growth appeared to be the largest contributor to the variation in estimated changes in the future health effects of air pollution.

This thesis provides a systematic investigation of the health risks of air pollution under the effects of future climate change, including a careful analysis of the sensitivity of the results to uncertainties. Assessing the health impacts of extreme air pollution episodes under climate change is an important area, not examined here, that should be the focus of future studies.

Contribution declaration for publication

Chapter 7 of this thesis is a published article in a peer-reviewed journal.

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I affirm that I was the lead author and took primary responsibility for overall management of producing this article. The table below shows approximate percentages of my contribution to this article.

Aspect of contribution	Percentage
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Each author of this article certifies that the level of contribution by the candidate indicated above is correct and grants permission for this paper to be included in the candidate's thesis.

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COMPONET 2

List of Abbreviations

AAQ NEPM	Ambient Air Quality National Environmental Protection Measures
ABS	Australian Bureau of Statistics
ACS	American Cancer Society
AIC	Akaike's Information Criterion
ANOVA	Analysis of Variance
APHEA	Air Pollution and Health: A European Approach
AQCD	Air Quality Criteria Document
ASGC	Australian Standard Geographical Classification
Avg	Average
AWS	Automatic Weather Stations
BoM	Bureau of Meteorology
Bsp	Particulate Measured by Nephelometry
CART	Classification and Regression Trees
CI	Confidence Interval
CMAQ	Community Multiscale Air Quality
CO	Carbon Monoxide
COPD	Chronic Obstructive Pulmonary Disease
CSIRO	Commonwealth Scientific and Industrial Research Organisation

CVD	Cardiovascular Disease
Df	Degree of Freedom
DOW	Day of the Week
ED	Emergency Department
EPA	Environmental Protection Authority
EPHC	Environmental Protection and Heritage Council
FB	Fractional Bias
FE	Fractional Error
GAM	Generalised Additive Model
GCM	General Circulation Model
GE	Gross Error
GHG	Greenhouse Gases
I/O	Indoor/Outdoor
ICD-10	International Classification of Diseases Version 10
IHD	Ischemic Heart Disease
IOA	Index of Agreement
IPCC	International Panel on Climate Change
IQR	Interquartile Range
Lowess	Locally Weighted Scatter Plot Smoothing
LR	Likelihood Ratio

Max	Maximum
MB	Mean Bias
Min	Minimum
ML	Maximum Likelihood
MLF	Most Likely Future
NCEPH	National Centre for Epidemiology and Population Health
NEPM	National Environmental Protection Measures
NMMAPS	National Morbidity, Mortality and Air Pollution Study
NO_2	Nitrogen Dioxide
NO_x	Nitrogen Oxides
O_3	ozone
PAPA	Public Health and Air Pollution in Asia
PM	Particulate Matter
PM_{10}	Particulate Matter with Diameter 10 Micrometres or Less
$PM_{2.5}$	Particulate Matter with Diameter 2.5 Micrometres or Less
ppb	Part Per Billion
pphm	Part Per Hundred Million
ppm	Part Per Million
PPR	Port Phillip Region
R	Correlation

RCM	Regional Climate Model
RD	Respiratory Disease
RMSE	Root Mean Square Error
SD	Statistical Division
SE	Standard Error
SLA	Statistical Local Area
SO_2	Sulphur Dioxide
SPIRT	Strategic Partnerships with Industry – Research and Training
SRES	Special Report on Emissions Scenarios
TAPM-CTM	The Air Pollution Model—Chemical Transport Model
TEOM	Tapered Element Oscillating Microbalance
USEPA	United States Environmental Protection Agency
VOC	Volatile Organic Compounds

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Chapter 1 Introduction

1.1 Background

With rapid industrialisation and urbanisation, air pollution has become one of the most important environmental risk factors posing a threat to human health. It has been estimated that there were approximately 3.24 million premature deaths worldwide in 2010 from exposure to ambient particulate matter (PM) and tropospheric ozone (O_3) air pollution (Lim et al. 2012). The major contributions to the premature deaths estimated were from the developing countries of Asia. The estimation has placed exposure to outdoor air pollution among the top 10 risk factors for loss of health in the 2010 Global Burden of Disease study. It is the responsibility of every government to protect and enhance air quality so that it is sufficiently clean to minimise adverse effects on public health. This may be achieved through establishing air quality standards and implementing regulations, policies and measures in order to bring the pollution under control. Although many countries have made good progress in improving their air quality, other challenges, including increases in urban populations, population ageing and the high incidence of non-communicable diseases, may offset efforts in air quality management by increasing the size of the populations at risk in the decades to come.

By their nature, meteorological conditions determine the quality of ambient outdoor air through atmospheric processes including emissions, chemistry, transport and deposition. Hence, the change of climate patterns that has been predicted in this century due to anthropogenic greenhouse gas (GHG) emissions will lead inevitably to alterations of the ambient air quality. Past studies have indicated that PM and O_3 are among the key pollutants of concern that are likely to be affected by climate change (Patrick and Kinney 2008; Jacob and Winner 2009). Most studies predicting O_3 have shown consistent findings of increases in future regional O_3 levels, particularly in polluted areas, while the direction and magnitude of changes in PM levels have been mixed among studies investigating the impact of climate change on future PM. This is mainly because temperature, which has been predicted to increase due to climate change, is the main meteorological parameter determining O_3 levels. For PM, because it comprises various components that respond to meteorological parameters differently, the predictions are more variable than for O_3. A few studies have found that precipitation was a key meteorological factor influencing future $PM_{2.5}$ concentrations (PM with an aerodynamic diameter of 2.5 micrometres or less) (Tagaris et al. 2007; Avise et al. 2009). Locations where precipitation levels are likely to be lower under a warming climate may have higher PM concentrations in the future.

Climate change affects air pollution and air pollution affects health. Thus, there is a need to project future health outcomes related to air pollution under climate change. Such projections can be made, based on assumptions on future scenarios of GHG emissions, air pollution emissions and population health, by linking estimated health risks of air pollution derived from epidemiological studies with the output of integrated climate and air quality models. Findings from past studies have shown increases in mortality and morbidity due to future elevated O_3 as a result of a warmer climate (Bell et al. 2007a; Chang et al. 2010; Cope et al. 2011a; Knowlton et al. 2008; Knowlton et al. 2004; Jackson et al. 2010; Tagaris et al. 2009). Because studies on projections of future PM concentrations responding to climate change have still been minimal, evidence on the associated health impacts of future PM levels has been sparsely reported (Ebi and McGregor 2008).

In Australia, there have been improvements in ambient levels of some air pollutants over the past decade (Department of Sustainability 2011; Barnett 2012). Levels of carbon monoxide (CO), nitrogen dioxide (NO_2) and sulphur dioxide (SO_2) monitored in major capitals of the country have seldom exceeded the Ambient Air Quality NEPM (National Environmental Protection Measures) standards. However, O_3 and PM remain of concern in air quality management and public health spheres. Breaches of levels of O_3 over the established goals and standards have been found regularly over the past decade in Sydney and occasionally in Melbourne. Exceedences of PM concentrations have occurred mainly in association with bushfire and dust storm episodes. No clear trends in O_3 levels over the past decade have been observed in Australia or in Europe. However, in the US, a decreasing trend, 13% lower levels of atmospheric O_3 in 2010 relative to 2001, has been reported (American Lung Association 2012). For PM concentrations, no clear trends over the last decade have been reported in Australia, whereas slight declines and moderate reductions were found in Europe and the US respectively (American Lung Association 2012; European Environment Agency 2012).

Even with the good air quality, air pollution is still identified as one of the environmental hazards posing a threat to Australians (Australian Institute of Health and Welfare 2011). Adverse health effects associated with air pollution have been repeatedly reported (Environmental Protection and Heritage Council 2010; Simpson et al. 2005a; Simpson et al. 2005b; Morgan et al. 1998b; Morgan et al. 1998a). An array of health outcomes, including effects on cardiovascular and respiratory disease and adverse birth outcomes (e.g., low birth weight, preterm birth, small of gestation age births and infant mortality),

have been associated with exposure to outdoor air pollution (Barnett et al. 2011; Hansen et al. 2012; Hansen et al. 2009; Cook et al. 2011; Hinwood et al. 2006). The evidence from these air pollution and health studies suggests that extra efforts from the federal and state governments to further control ambient air pollution and to identify other preventive measures to ameliorate the adverse health effects in the Australian population are still needed.

Changes in climate projected in the decades to come in Australia will have direct and indirect impacts on health (Saniotis and Irvine 2010; Woodruff et al. 2006). Potentially, the health outcomes associated with air pollution may be modulated under a changing climate through two key pathways: i) increases in intensity and frequency of extreme events, including heatwaves and bushfires, and ii) increases in the number of hot days.

Extreme weather events, particularly heatwaves and bushfires, are expected to become more common under a future warming climate, and have the potential to reduce air quality, resulting in adverse health consequences. In recent years, an increased frequency and intensity of these two events has been clearly evident in many Australian regions (Hughes and Steffen 2013). Epidemiological studies on the 2003 European heatwaves have provided information on the contribution of increased O_3 levels to excess deaths in addition to heat exposure (Lacour et al. 2006; Vautard et al. 2007; Fischer et al. 2008). A recent study estimated an excess of 3 200 deaths due to exposure to PM_{10} and O_3 related to wildfires and interactions between temperature and PM_{10} in Moscow, Russia, over a period of 44 days of the heatwave in 2010 (Shaposhnikov et al.). In Australia, in part due to a lack of definition of heatwaves and also because the magnitude of adverse health effects on very hot days has not been as severe as the 2003 heatwave in Europe, investigation of air quality and related health effects during heatwaves is still minimal. Nevertheless, a study that estimated excess deaths during the 2004 heatwaves in Brisbane found that exposure to maximum O_3 concentrations induced approximately 20% of excess deaths for non-external and cardiovascular causes during that period, whereas the other 80% of the excess deaths estimated was attributable to exposure to maximum temperature (Tong et al. 2010b).

Bushfires, severe events triggered by high temperatures and extended droughts have caused direct fatalities and injuries from flame exposure and indirect health burdens in relation to fire smoke inhalation in Australia. Air pollution levels during bushfires can dramatically exceed the safety levels. For example, readings from air quality monitoring

sites during the 2009 Black Saturday bushfires in Victoria, the most severe bushfires in Australia, showed PM_{10} (PM with a diameter of 10 micrometres or less) concentrations three times greater than the national standards (EPA Victoria 2009). A number of studies across major cities in Australia have found evidence of associations between health effects and exposure to air pollution during bushfires events (Chen et al. 2006; Johnston et al. 2011; Tham et al. 2009).

An increase in the number of hot days and elevated temperatures projected in this century as a result of climate change may also alter future air quality and related health consequences. One potential pathway is through changes in the formation and emission of air pollutants sensitive to temperature as mentioned above. Another possible mechanism might be associated with interaction effects of temperature and air pollution on health. Findings from a number of studies suggest that mortality and morbidity risks associated with air pollution are variable across temperature ranges. Studies from overseas found greater health risks of air pollution at extreme cold and hot temperatures, depending on the climate conditions of where the research was conducted (Roberts 2004; Carder et al. 2008; Cheng and Kan 2012). Although only a few studies have been conducted in Australia, similar findings from a study in Brisbane were also reported, showing stronger health risks of air pollution at high temperature extremes (Ren and Tong 2006).

1.2 Statement of the problem and motivation of the study

To date, information needed to aid long-term air quality management and the development of necessary responses to protect populations from changes in future air pollution induced by climate change is lacking. Health impacts assessment through future projections is a key tool to provide such information. However, science in this area is still at a developmental stage. This can be seen from the limited number of studies attempting to project health impacts from future changes in air pollution due to climate change (Sujaritpong et al. 2013).

In the research area of the impact of climate change on health effects associated with air pollution, three key priorities are needed to progress further. The first priority is to make projections of future air quality and its consequences for health at regional and local levels. Most studies in the past have focused on projecting the impacts at global and continental scales, in part due to constraints in developing future climate projections at high resolution. Lack of information from the projections at regional and local levels is problematic for

developing policies and control measures on the ground that address the needs at specific locations with different circumstances in air quality and population health.

The second priority concerns uncertainties around assumptions made in the projections. Addressing uncertainties associated with future GHGs and climate modelling is common and the knowledge in this area is well established. However, other uncertainties, including future air pollution emissions, changes in future demographic features, population health scenarios and the choice of relative risks for health outcomes associated with air pollution, are important, but have been largely overlooked. Some of these uncertainties can have a great deal of impact on the projection results, which are even greater than the uncertainties associated with GHGs and climate modelling (Kolstad and Johansson 2011). Incorporating these uncertainties into projections exercises and reporting consequent estimates of the health impacts transparently could provide more useful information in decision making processes.

The third priority is to make more efforts in projecting future PM concentrations and estimating relevant health effects. Given the current improvement of climate projections at fine scales, there will be more opportunities in advancing future PM projections and providing more accurate future PM concentrations, since PM is highly variable with locations and local meteorology, compared with O_3.

Studying the interaction effects of temperature and air pollution on health through epidemiological approaches has provided critical knowledge in understanding health impacts associated with air pollution under a changing climate. However, the knowledge derived and dose-response relationships estimated from these studies have not yet been linked to the mainstream process of assessing future health impacts from air pollution and climate change. Therefore, it is important in the next step in this research area to explore the extent to which dose-response relationships gained from epidemiological studies that consider the interaction effects of temperature and air pollution can make the future projections of health impacts more informative; assuming that such interaction effects exist and are influential. Additionally, the use of such dose-response relationships can also provide an insight with regard to the sensitivity of the future health impacts to dose-response relationships derived from different methods and assumptions.

Past studies that have predicted future air quality under a changing climate have used atmospheric chemical transport models. The fine resolution output from such models facilitates detailed simulation across an air shed of interest. In contrast, dose-response relationships derived to predict the future health impacts of air pollution have largely used measures of ambient air quality obtained from monitoring networks, usually aggregated to the city level. This mismatch of the spatial scale of the air pollution estimates versus these dose-response relationships may limit the accuracy and/or precision of estimates of future health risks related to air pollution.

Responding to the concerns mentioned above, alternatively, an air quality model can be used to simulate air pollution exposures for estimating a dose-response function for the purpose of future health impacts assessment. Predictions based on air quality modelling have been applied in epidemiological studies on air pollution and health effects and have proved to be beneficial in several aspects, for example, addressing issues associated with measurement errors and investigating possible spatial variation on health effects attributable to air pollution at different scales (Erbas et al. 2005; Bell 2006; Valari et al. 2011). Details of such a benefit will be elaborated in Section 2.4.1. However results derived from applying air quality modelling for studying the relationships between air pollution and health have not yet extended to the projections of future health impacts. With the consistent methods in estimating air pollution exposures for estimating dose-response functions and for predicting changes in future air quality, projections of the future health impacts could potentially be made in a more precise and reliable fashion. There is a need, therefore, to address the concern of using air quality modelling in estimating health risks of air pollution for the improvement of future projections in future research.

As for the Australian context, similar to elsewhere, assessing climate change impacts on air quality and associated health consequences is still a new area of research and further development is necessary. Two studies have attempted to project changes in air quality in response to future climate change in Australia (EPA Victoria and CSIRO 2013; Cope et al. 2008b). The first study was conducted primarily to demonstrate methods for assessing the impact of climate change on O_3 at a city level by using the setting of the Sydney Region as an example. In that study, future respiratory hospital admissions in relation to an increase of O_3 concentrations projected to the 2020s and 2050s were estimated. Since the aim of that study was not to truly estimate the air pollution health outcomes, simplified scenarios on key drivers impacting future changes in O_3 levels and health consequences were

assumed and critical uncertainties such as those associated with climate modelling were not considered. Clearly, if a real assessment on potential health impacts of future air quality related to climate change is to be made, more complete future scenarios must be taken into account.

Another study in Australia, known as the Future Air Projections project (referred to as 'the project' in this thesis), aiming to project future air quality in the Port Phillip Region (PPR)—Melbourne and Geelong—, Victoria, was started in 2009 and completed in 2011(EPA Victoria and CSIRO 2013). Some of the methods employed in projecting future air quality were similar to the previous study. However, the study in the PPR added several dimensions that differed from the previous study and were critical in making future air quality projections. Air pollutants considered in that project were not limited to only O_3; other pollutants including PM were also studied. Important factors likely to affect future air pollution in the region, which were not considered in the previous study, including changes in GHG emissions, air pollution emissions and population, were thoroughly investigated. Given the comprehensive assessment of future air quality undertaken by the project, building on its work to further analyse consequences in public health enables advancing knowledge in the research area of future health impacts associated with air pollution due to climate change. In particular, the identified research gaps and the questions around the accurate projection of future health impacts aforementioned can be further explored.

1.3 Research aim, questions and objectives

The aim of this thesis is to assess the impact of future climate change on the health effects of air pollution in the Melbourne Region. As identified in the previous section of this chapter, there are a number of gaps and questions for the further progress of assessing the future health impacts of air pollution. In this thesis, to address the research gaps and questions in order to ultimately provide credible projections results, the following research objectives are set as shown in Table 1.1.

1.4 Research framework

This thesis is framed under two major components as illustrated in Figure 1.1. Using retrospective datasets for the period 1999 to 2008, Component 1 involves addressing Objectives 1 and 2 in estimating baseline relative risks for acute health outcomes

associated with air pollution under the influence of interactions between air pollution and temperature in the Melbourne Region. Component 2 aims at addressing Objectives 3 and 4 in order to assess future health impacts of air pollution, taking into consideration various aspects of uncertainties.

Table 1.1 Research questions and corresponding research objectives

Research question	Research objective
	Objective 1
What is the best approach for the purposes of estimating relative risks for acute health outcomes associated with air pollution and assessing the impact of future climate change on the health effects in the Melbourne Region?	To identify the best approach for estimating relative risks for acute health outcomes associated with air pollution and assessing the impact of future climate change on the health effects in the Melbourne Region.
	Objective 2
For the Melbourne Region over the baseline period 1999 to 2008: Are there any interaction effects between temperature and air pollution on acute health outcomes? If so, to what extent do the interaction effects modify relative risks for acute health outcomes associated with air pollution?	For the Melbourne Region over the baseline period 1999 to 2008: To explore interaction effects between temperature and air pollution on acute health outcomes. To estimate relative risks for acute health outcomes associated with air pollution, taking into account the interaction effects of temperature and air pollution.
	Objective 3
To what extent will future air quality be modified by climate change impact on acute health effects associated with air pollution in the PPR, including when the interaction effects of temperature and air pollution are taken into account?	To estimate changes in the acute health effects of air pollution due to future changes in air quality modified by climate change in the PPR for the period 2065 to 2074, including when the interaction effects of temperature and air pollution are taken into account.
	Objective 4
To what extent will non-climate factors and projection methods associated with the health impact projections affect the estimated changes in the acute health effects of air pollution undertaken in Objective 3?	To explore the sensitivity of estimated changes in the acute health effects of air pollution undertaken in Objective 3 to non-climate factors and projection methods.

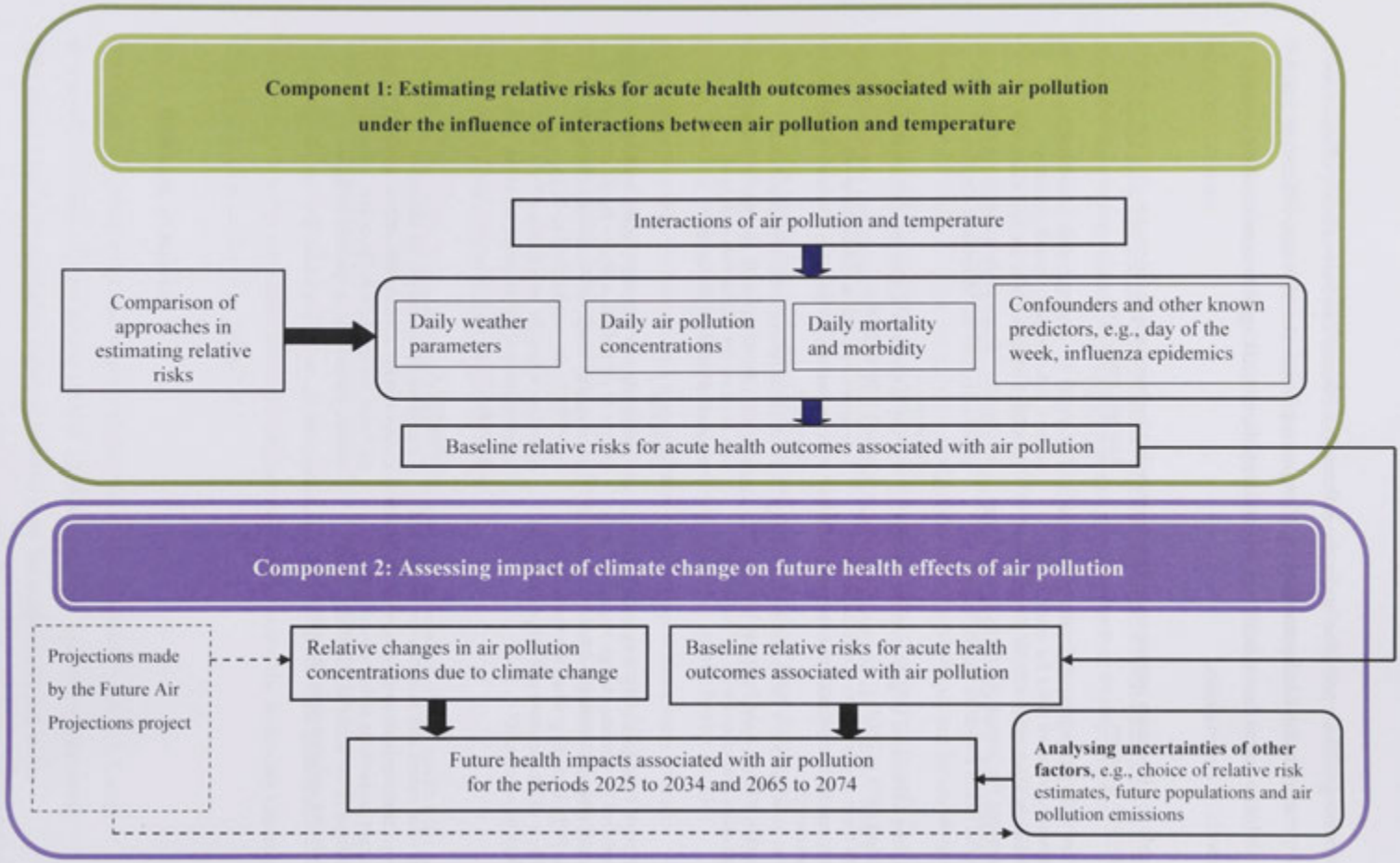


Figure 1.1 Research framework

Component 1 involves combining four main datasets—daily mortality and morbidity, daily air pollution concentrations, daily weather parameters and information on potential confounders—to estimate relative risks for acute health outcomes associated with air pollution for the reference period 1999 to 2008. The research questions forming Objectives 1 and 2 are addressed through examining estimates of the relative risks under this component.

The research question corresponding to Objective 1 as shown in Table 1.1 addresses the uncertainty in the best method to estimate air pollution exposures when quantifying the associations with health outcomes and assessing future health impacts as described earlier in Section 1.2. Objective 1 is attained by comparing the results from four different approaches for estimating exposure to air pollution on a range of health outcomes. The standard approach in time-series studies of health and air pollution gained from averaging measurements of fixed monitoring stations to provide air pollution exposures for an entire city was benchmarked against alternative approaches. Among the alternative approaches chosen to compare here, one used an air quality modelling system, The Air Pollution Model—Chemical Transport Model (TAPM-CTM), to estimate air pollution concentrations and weather data. The reason to choose this modelling system is that it was the system employed in predicting changes in future air quality under a changing climate in the PPR for the Future Air Projections project.

Built on the best approach for estimating the relative risks selected in Objective 1, this thesis addresses Objective 2 by subsequently stratifying temperatures into different strata in order to: i) explore the existence of the interaction effects of temperature and air pollution on the health effects of air pollution and ii) quantify relative risk estimates of the health effects of air pollution for certain temperature strata.

The interaction effects of temperature and air pollution on acute health outcomes associated with air pollution have not been considered in past studies projecting future health impacts from climate change. This thesis, hence, explores the degree to which taking such interaction effects into consideration is important in estimating the impact of climate change on future health risks associated with air pollution, which is Objective 3, embraced in Component 2. The relative risks estimated from Component 1 are linked with relative changes in air pollution concentrations between the baseline period 1996 to 2005 and a future time window 2065 to 2074 in the PPR, predicted by the project for the

estimation. Equation 1.1 is a standard formula used in estimating health impacts of changes in an environmental factor on population health:

$$\Delta H = R\left(e^{\beta \Delta C} - 1\right)Pop, \quad (1.1)$$

where ΔH is the change in the health outcome of interest resulting from changes in an environmental factor, R is the baseline mortality or morbidity rate, ΔC is the estimated change in an environmental factor, β is the log relative risk associated with a change in exposure to the environmental factor and Pop is the exposed population in the period and location of interest. It can be seen that additional information on future demographic features R and Pop is also required apart from the baseline relative risks (β) and the relative changes in air pollution concentrations (ΔC). However, under this objective, the focus was on the impact of climate change. Therefore, the estimation for this objective was made on the basis of assuming only changes in GHG emissions according to one high emissions scenario—the A2 SRES—developed by the International Panel on Climate Change (IPCC). In the Future Air Projections project, this assumption was referred to as 'Climate Penalty' (EPA Victoria and CSIRO 2013). Other factors than the change in GHG emissions under the 'Climate Penalty' scenario were assumed to be constant over future periods to emphasise the impact of climate change on air pollution and its consequences on health.

Given that the estimates of the future health impacts predicted in Objective 3 may be sensitive to other factors, Objective 4 was set to explore this aspect of the future health impacts assessment. The factors considered under Objective 4 included those that might significantly affect the components expressed in Equation 1.1—the choice of relative risks, changes in future air pollution emissions determining ambient air pollution levels and demographic changes of future populations.

1.5 Research scope and limitations

1.5.1 Study area

Given the advanced computer technology and ability of current climate modelling systems in downscaling to urban resolutions, the scale of interest of this thesis is at the city level. The Melbourne Metropolitan Region as shown in Figure 1.2 was chosen as the area of

study for three main reasons, including its population size, levels of air pollution in the city and availability of predictions of future air quality in the Melbourne Region.

Figure 1.2 Study area in the Melbourne Metropolitan Region, Victoria

Source: adapted from
http://upload.wikimedia.org/wikipedia/commons/8/8c/Melbourne_Map.png

Melbourne has the second largest population in the country. The population of the Melbourne Region, based on the 2011 Census, was 4.1 million (Victorian Department of Planning and Community Development 2012). On the one hand, the large population in Melbourne and the related anthropogenic air pollution emitted are among the significant contributors that aggravate its air quality. On the other hand, the larger the population is, the greater the public health concern because of the large number of people exposed to air pollution. Although the air quality in Melbourne is considered generally good, the level of air pollution in Melbourne, based on annual average levels of O_3 and PM_{10} during 2000 to 2005, compared to other Australian metropolitan cities, was ranked as the second poorest, inferior only to Sydney (Department of Sustainability 2011).

Availability of predictions of future air quality in the Melbourne Region under the Future Air Projections project was the other important reason for studying this region. The predictions made from the project provided opportunities to further investigate health consequences resulting from future changes in air quality due to climate changes as well as other influences.

The boundary of the study area is limited at Statistical Division (SD) code 205 of the Australian Standard Geographical Classification (ASGC), which includes 79 Statistical Local Areas (SLAs) as shown in Figure 1.3.

1.5.2 Periods of interest

The baseline relative health risks associated with exposure to $PM_{2.5}$ and O_3 without and with inclusion of effect modification of temperature to address Objectives 1 and 2 respectively in this thesis are estimated based on datasets in the period 1999 to 2008.

For Objective 3, this thesis compares a baseline period 1996 to 2005 with a period centred on 2070 (2065 to 2074). For Objective 4, a future period centres on 2030 (2025 to 2034) with a comparison of the same baseline period. The 2030 period represents the short-term future, while the 2070 period reflects a timeframe for the medium-term future. The 2030 period was specifically chosen because there have been projections for other purposes in the state of Victoria focusing on this timeframe, in particular for demography, and regional planning and development (Victorian Department of Planning and Community Development 2012). Thus, the use of such a timeframe allowed linking the estimates of health impacts made in this thesis to other relevant projections available.

Figure 1.3 The boundary of the study area (SD code 205) and its division into 79 SLAs
Source: adapted from Jacki (2010)

1.5.3 Air pollutants of interest

Due to urban sprawl of the Melbourne Region, the major source of air pollution in the city is motor vehicle emissions. Apart from the anthropogenic sources, natural sources are important causes of air pollution episodes, especially pollutants during bushfires and dust storms in the Melbourne Region. With the success of air quality management over the past decade, air pollutants mainly emitted from motor vehicles, including CO, Nitrogen oxides (NO_x) and SO_2, have been largely brought under control (Department of Sustainability 2011). The exceptions are for the two pollutants, PM and O_3, which are still of concern nowadays; these were the pollutants of interest in this thesis. For PM, this thesis focuses only on $PM_{2.5}$, which is more harmful to health than the coarser fractions of PM (Brook et al. 2010; Martinelli et al. 2013).

As stated earlier, this thesis used the future air pollution concentrations predicted by the Future Air Projections project in estimating the impact of climate change on the health effects of air pollution. The Future Air Projections project did not include future emissions

from bushfires and dust storms into TAPM-CTM runs in order to predict changes in future air quality in the PPR (EPA Victoria and CSIRO 2013). This thesis, thus, limits the future projections of the health effects attributable to air pollution on day-to-day circumstances and disregards the health impacts associated with exposure to air pollution during extreme air pollution events.

To be consistent with the future projections of air pollution-related health effects under climate change, estimating baseline relative risks in this thesis was limited to only days in which the air quality in the Melbourne Region was not considerably affected by emissions from bushfires and dust storms. To do so, $PM_{2.5}$ and O_3 concentrations derived from observations and simulating the air quality modelling system on days in which the exceedances of PM_{10}, $PM_{2.5}$ and O_3 were reported and bushfires were identified by EPA Victoria to be inferred causes were not considered. Similarly, days on which dust storms were inferred to be causes of the exceedances of $PM_{2.5}$ and PM_{10}, $PM_{2.5}$ concentrations were not considered.

From a perspective of modelling the relationship between air pollution and health, there are two additional reasons for limiting estimations of the baseline relative risks only to days without the impacts of extreme air pollution episodes. First, removing days with unusually high values of air pollution concentrations has been widely applied in large-scale studies investigating the health effects of air pollution. This is to ensure that relative risk estimates are not biased by a few major extreme events (Simpson et al. 2005b). Second, isolations of days with extreme air pollution episodes in this thesis is based on some recent evidence shown in studies investigating health risks associated with different sources and components of $PM_{2.5}$. These studies suggest that PM emitted from different sources and components impact variously on health (Martinelli et al. 2013). This is due to the difference in PM chemical components corresponding to each source. PM-related industrial and traffic sources are commonly rich in heavy metals such as nickel and arsenic. Hence, health risks related to these two sources have been found to be greater than those from other sources (Zanobetti et al. 2009; Laden et al. 2000).

Considering the evidence above, it is possible that risk estimates associated with $PM_{2.5}$ in the Melbourne Region on normal days may differ from those when extreme air pollution episodes occur. A study that investigated sources of ambient PM in four major cities of Australia concluded that $PM_{2.5}$ was emitted mainly from eight major sources (Chan et al.

2008). Emissions from motor vehicles accounted for 24%, making it the largest source for the ambient $PM_{2.5}$. Emissions from bushfires, controlled burnings and domestic wood heating that were classified into another category referred to as 'other combustion sources' generally accounted for only 8%. During extreme air pollution episodes, these contributory percentages would certainly change. For instance, during bushfires, the 'other combustion sources' could become dominant and modify the percentages of source contribution. This could result in altered risk estimates. The extent to which the use of datasets with and without extreme air pollution episodes has an impact on the health risk estimates associated with $PM_{2.5}$ is explored and discussed in Chapter 5.

1.5.4 Health outcomes investigated

Health outcomes in this thesis were restricted to mortality and emergency department (ED) visits of Melbourne residents for respiratory and cardiovascular disease. All the respiratory diseases classified under the respiratory category (J00-J99) according to the International Classification of Diseases version 10 (ICD-10) were combined to form a large group of respiratory disease for the purpose of estimating the relative health risks. The same was done with the cardiovascular disease category (I00-I99). All age groups were included, although many studies indicate that children and the elderly are particularly sensitive groups.

1.6 Thesis structure and overview

This thesis is organised into four main sections. The first section, ***Chapter 1***, provides the background and rationale for this thesis. It also provides a framework outlining an overall picture of this thesis. The scope and limitations of this thesis are clarified in this chapter to set out the boundary for the following chapters.

The second section, ***Chapters 2–6***, addresses Objectives 1 and 2, focusing on estimating relative risks for acute health outcomes associated with air pollution, taking into account the interaction effects of temperature and air pollution. ***Chapter 2*** functions as a review of three topics: i) the development of epidemiological studies of air pollution and health worldwide generally and in Australia specifically, ii) past studies examining the interaction effects of temperature and air pollution on health and iii) roles of air quality modelling in epidemiological studies of air pollution. ***Chapter 3*** describes datasets used to estimate relative risks of the acute health outcomes for the baseline period 1999 to 2008. The

datasets included in this chapter are daily health outcomes and observations of the air pollutants and weather parameters considered. ***Chapter 4*** describes an air quality modelling system employed to generate modelled air pollution and weather data, and its performance compared to the observations. This chapter also presents a technique adopted to blend the modelled datasets with the observations. Similar to the performance of modelled data evaluated against observations, the datasets generated from the blending technique were also evaluated. ***Chapter 5*** explains statistical methods and criteria used in choosing four approaches based on the datasets of air pollution and weather described earlier in Chapters 3 and 4. The selected approach was used further to model relative risks of air pollution under the influence of interactions between air pollution and temperature. ***Chapter 6*** describes statistical models used based on the selected approach in estimating relative risks associated with air pollution modified by temperature. This chapter concludes by discussing how the estimated health risks taking into account the temperature modifying effect in this chapter were used in the following steps for the future projections.

The third section, ***Chapters 7 and 8***, centres on the assessment of future health impacts associated with future air pollution under a changing climate, which addresses Objectives 3 and 4. ***Chapter 7*** is a published article in a peered-review journal that serves as a review of methods used by past studies projecting the future health impacts (Sujaritpong et al. 2013). ***Chapter 8*** explains briefly the Future Air Projections project and how and which elements of its output were used in this thesis. It is then followed by methods to undertake future projections of the health impacts of air pollution and shows how the results from previous chapters were combined in the projection processes. It also shows how uncertainties for each step of the projections were considered and reported as part of uncertainty analyses. The projected results are presented and discussed at the end of this chapter.

Chapter 9 is the last section of this thesis. It weaves the previous three sections together. It starts with recapping the aim of this thesis and summarising the key research needs identified and addressed by this thesis as stated in Chapter 1. It summarises findings from the second and third sections to address the aim, questions and objectives of this thesis. It analyses strengths and limitations of this thesis and recommends how to apply the thesis findings in the policy arenas for managing air quality and protecting public health in the future under a changing climate. It ends by identifying remaining gaps in this research area.

COMPONENT 1

Estimating Relative Risks for Acute Health Outcomes Associated with Air Pollution under the Influence of Interaction between Air Pollution and Temperature

Chapter 2 Literature review

2.1 Introduction

To achieve the research aim of this thesis, one of the objectives, as described in the previous chapter, involves developing a dose-response function for the health outcomes of interest induced by air pollution. As this study focuses on the health impacts of air pollution in Melbourne, Australia, this chapter begins by reviewing previous epidemiological studies estimating health risks of air pollution in major capital cities of Australia. The second part of this chapter—the main part—is a review of epidemiological studies that have taken into account interaction effects of temperature and air pollution on health. The existence and patterns of the interaction effects as well as variations of findings in these studies are examined and discussed. Importantly, the methods applied in these studies are examined to choose approaches that are suitable in estimating relative health risks associated with air pollution exposure modified by temperature in Chapter 6. Plausible mechanisms of the interaction effects that could be used to explain findings from these studies are explored and summarised. As outlined in Chapter 1, one of the approaches examined in this thesis for estimating health risks associated with air pollution exposure involves using air pollution and weather data simulated by an air quality and meteorological modelling system, TAPM-CTM. With this regard, the last section includes a discussion about major roles of air quality modelling in air pollution and health studies.

2.2 Epidemiological studies examining health risks associated with air pollution

2.2.1 General background

The importance of epidemiological studies examining air pollution-related health risks has been well recognised since the initial discoveries of adverse health effects caused by air pollution episodes in the last century (Wichmann et al. 1989; Davis 2002; Mark Z. Jacobson 2002). Historically, among important air pollution episodes occurring in Europe and North America in the 19th and 20th centuries, the episode in London in December 1952 is the most well-known due to its severity. For that air pollution episode, it was estimated that there were approximately 12 000 deaths resulting from exposure to the fog containing high levels of SO_2 and PM emitted from coal combustion (Davis 2002). Out of the total number of deaths, 4 000 occurred during the event, while the remainder were thought to be due to a delayed effect of the exposure. Similar air pollution episodes caused by a mixture of fog and smoke from air pollution trapped near the ground due to temperature inversions have been referred to as 'London-type smog'.

Evidence from these air pollution events—from past epidemiological studies, along with that from animal studies and human clinical studies—has been used to establish air quality standards and regulations, and to develop policies and strategies to limit harm on populations from air pollution. Apart from strong evidence on associations between cardiopulmonary disorders and air pollution, increasingly in recent years, research findings have shown an additional array of adverse health outcomes, notably abnormalities of birth outcomes (Rémy Slama et al. 2008) and nervous system effects (Fonken et al. 2011; Lucchini et al. 2011; Genc et al. 2012). In addition, toxic effects of air pollution resulting in DNA damage, which is likely to be a contributing factor of cancer development based on laboratory studies, have been also demonstrated in epidemiological studies (Wei et al. 2011; Moller et al. 2008). Therefore, the new scientific evidence generated from these on-going epidemiological studies is also necessary and beneficial in evaluating effectiveness of current standards, regulations and strategies and updating them as required.

Several epidemiological approaches have been used to estimate the health effects of air pollution. Based on study design, epidemiological studies of air pollution can be classified into four types: time-series, case-crossover, cohort and panel studies (Peng and Dominici 2008). Although each of these study designs has inherent advantages and disadvantages, they provide different types of evidence that can be used to mutually validate and support each other. Time-series and case-crossover studies can provide evidence of associations between acute air pollution exposure and broad disease classifications such as cardiovascular and respiratory diseases. However, such evidence cannot be used to pinpoint specific diseases and explain potential causal pathways. Panel studies can fulfil this role by providing specific evidence on changes in certain body functions to reveal suspected causal relationships between health endpoints and exposure to air pollution. For example, studying relationships between changes in heart rate variability and exposure to air pollution in panel studies (Bartell et al. 2013; Shields et al. 2013) can be used to confirm and explain increased cardiovascular outcomes found in time-series and case-crossover studies of the health effects of air pollution.

Among these study designs, the time-series approach has been applied broadly to investigate short-term health effects of air pollution (Ren and Tong 2008). Shortcomings of conventional methods of time-series studies applied to single-site studies, including insufficient statistical power to detect the health effects and a lack of heterogeneity of effect modifiers, have been debated widely in the research community. To address these

issues, several large time-series studies using a multi-site approach, for example, National Morbidity, Mortality and Air Pollution Study (NMMAPS) in the US (Peng and Dominici 2008), Air Pollution Health Effects in Europe: a European Approach (APHEA) in Europe and Public Health and Air Pollution in Asia (PAPA) (Wong et al. 2008), have been set up in different parts of the world over the last two decades. These large-scale studies have provided insightful findings, leading to the conclusion that short-term exposure to outdoor air pollution can induce increases in all-cause mortality, as well as cardiorespiratory mortality and morbidity.

To detect long-term health effects of air pollution, several cohort studies have been conducted. Examples of these studies include the American Cancer Society (ACS) study and Harvard Six-Cities study. These studies have reported increased risks of mortality and morbidity for cardiorespiratory diseases and lung cancers as a consequence of chronic exposure to outdoor air pollution (Pope 2007).

2.2.2 Previous studies in Australia

A number of epidemiological studies have been conducted to estimate health risks of exposure to outdoor air pollution in Australia. In the early stages of Australian epidemiological research on air pollution, most studies used the conventional time-series approach to estimate relative risks of the short-term health outcomes in single cities. These studies paid attention to the impact in large capital cities; Sydney, Melbourne, Brisbane and Perth (Morgan et al. 1998a; Morgan et al. 1998b; EPA Victoria 2000, 2001; Simpson et al. 1997). In view of the limitations (stated above) of the single-site approach, two national-scale studies have been conducted using a multi-site approach. The rationale for these two studies was to provide overall relative risk estimates for key short-term health endpoints that could be applicable nation-wide. A further value was as a source of scientific evidence for future reviews of National Environmental Protection Measures (NEPM) for ambient air quality (Simpson et al. 2005a; Simpson et al. 2005b; Environmental Protection and Heritage Council 2010). The first of these multi-site studies is commonly referred to as SPIRT (Strategic Partnerships with Industry – Research and Training) and the second, more recent, as EPHC (Environmental Protection and Heritage Council). The SPIRT study included four cities in Australia (Sydney, Melbourne, Brisbane and Perth), while the EPHC study included the four cities in the SPIRT study with the

addition of one city in Australia (Canberra) and two cities in New Zealand (Auckland and Christchurch).

2.2.2.1 Recent progress in studying the health effects of air pollution in Australia

Over the past decade, epidemiological research on air pollution in Australia has progressed substantially. Apart from the time-series approach, other study designs, including case-crossover (Jalaludin et al. 2008; Pereira et al. 2010), case-control (Cook et al. 2011; Hansen et al. 2009) and cohort studies (Pereira et al. 2012; Bennett et al. 2007), have been applied to investigate the short-term and long-term health effects of air pollution. Recent investigations have not been limited to large capital cities but have also included smaller capital cities. For example, a study has been conducted to examine cardiorespiratory hospital admissions related to PM in Adelaide, one of the capital cities not included in the two national studies (Hansen et al. 2012). In Darwin, the capital city of the Northern Territory, where air pollution from anthropogenic sources is minimal, there have been a number of studies investigating health effects from exposure to bushfire smoke (Hanigan et al. 2008; Johnston et al. 2007; Johnston et al. 2002).

In addition to an expansion in the types of studies undertaken, the range of health outcomes has been extended to now include birth outcomes (Barnett et al. 2011; Mannes et al. 2005; Pereira et al. 2011). Further, the health consequences of interactions between temperature and air pollution, an issue that emerges from consideration of the potential impacts of climate change, has also been researched (Ren and Tong 2006; Hu et al. 2008; Ren et al. 2006). Details about this research area in Australia are elaborated in the following section.

2.2.2.2 Summary of findings from epidemiological studies relating adverse health effects with outdoor air pollution exposure in Australia

As the main focus of this thesis is on short-term health endpoints, a review of findings of previous epidemiological studies of air pollution is limited to associations between daily changes in health outcomes and outdoor air pollution concentrations. A systemic literature search for Australia studies was conducted, using PubMed and Web of Science, and the following criteria: (a) key words: air pollution, PM, ozone, health, mortality, Australia, Adelaide, Brisbane, Perth, Melbourne and Sydney; (b) studies published before 1996; and (c) studies applied time-series and case crossover approaches. In addition, all the studied cited by two reports – "Expansion of the multi-city mortality and morbidity study"

(Environmental Protection and Heritage Council 2010) and "Monitoring the Impact of Air pollution on Asthma in Australia" (Australian Institute of Health and Welfare 2010) – were included in the review. Key findings of the reviewed studies are presented here and summarised in Tables 2.1 and 2.2 for mortality and morbidity respectively. Note that studies that examine the potential health effects from exposure to bushfires and dust storms are excluded from this review.

Air pollutants of interest in relation to short-term health outcomes in Australia include CO, Bsp, NO_2, PM_{10}, $PM_{2.5}$, O_3 and SO_2. Bsp, a measurement of visibility undertaken using nephelometry, has occasionally been used as a surrogate for PM in these studies. With the high correlation between Bsp and $PM_{2.5}$, a number of studies used Bsp data to investigate health effects due to exposure to $PM_{2.5}$ in the early 1990s, when an instrument to measure this pollutant was not yet available. SO_2 and CO were excluded from analysis in some studies (e.g. Simpson et al. 2000; Morgan et al. 1998a; Environmental Protection and Heritage Council 2010; Simpson et al. 2005a) as their concentrations have been continuously low over the past decade (Department of Sustainability 2011).

Major health endpoints of concern have been within broad categories of all-cause, cardiovascular and respiratory diseases. Some studies further examined specific health outcomes believed to have biologically causal relationships with exposure to PM, for example, stroke, ischemic heart disease (IHD), asthma and chronic obstructive pulmonary disease (COPD). Stratification by age and season has been a common approach in these studies.

2.2.2.3 Mortality outcomes associated with air pollution in Australia

Using the single-site approach, significant associations have been found between mortality and acute exposure to several pollutants although the associations vary across the cities (Table 2.1). Three single-site studies in Brisbane (Simpson et al. 1997), Melbourne (Simpson et al. 2000) and Sydney (Morgan et al. 1998a) had consistent findings showing significant relationships between all-cause mortality and exposure to Bsp/PM and O_3. However, the results for NO_2 varied from city to city. A significant association between NO_2 and all-cause mortality was not apparent in Brisbane (Simpson et al. 1997) but was present in Melbourne and Sydney. Findings on relationships between air pollution and cardiovascular and respiratory mortality were also variable across these three cities. In Melbourne, there were no significant associations between cardiovascular mortality and

exposure to any air pollutants examined, while in Sydney no associations were evident to that between any pollutants and respiratory mortality. In Brisbane, exposure to Bsp was associated with increased cardiovascular mortality, but not respiratory mortality.

Although differences in the evidence for associations across these cities are apparent in these single-site studies, there is no significant heterogeneity between cities when tested within the two multi-site studies. Overall pooled relative risks estimated in the two multi-site studies confirmed the associations seen in the single-site studies for all-cause and specific mortality categories in association with Bsp/PM, NO_2 and O_3 (Environmental Protection and Heritage Council 2010; Simpson et al. 2005b). However, results of these two multi-site studies differed slightly for the associations between O_3 and mortality outcomes. In the SPIRT study (Simpson et al. 2005b), there was a strong association between exposure to O_3 and respiratory mortality. In contrast, the EPHC study (Environmental Protection and Heritage Council 2010) found significant associations between all-cause and cardiovascular mortality and exposure to O_3 , but not respiratory mortality. Such a difference may be partly due to the use of O_3 data between these two studies. The EPHC study restricted O_3 data to only warm months (November to April) whereas the SPIRT study used all-months O_3 data. Despite the difference in the mortality effects of O_3, based on making a comparison, it was concluded that overall there was no difference between the results derived from these two studies (EPHC 2010).

2.2.2.4 Morbidity outcomes associated with air pollution in Australia

More studies in Australia have investigated associations of air pollution with morbidity than with mortality. The studies summarised in Table 2.2 cover major Australian capitals. Daily morbidity data examined in these studies were derived from two sources, namely emergency hospital admissions and ED visits. Main disease categories of interest in these studies include broad cardiovascular and respiratory classifications. Due to the high incidence of childhood asthma in Australia in recent decades, some studies limited their scope of investigation only to an association between asthma and air pollution in children (Erbas et al. 2005; Pereira et al. 2010; Jalaludin et al. 2008). Overall, results from these studies show evidence of the effect of air pollution on various morbidity outcomes, even at ambient concentrations that were well below the NEPM ambient air quality guidelines.

Table 2.1 Studies in Australia on mortality associated with air pollution

Reference	Study location(s) and period	Method	Main findings*
Simpson et al. (1997)	Brisbane 1987–1993	Time-series	Relative risks for all-cause mortality in 65+ years group in all months: Bsp 24-h avg, lag0: 1.010 (95% Confidence Interval [CI]: 1.002, 1.018) per $10^{-5} m^{-1}$ O_3 max 1-h and 8-h, lag0: 1.016 (95% CI: 1.004, 1.028) and 1.024 (95% CI: 1.005, 1.042) per 10 ppb NO_2 and SO_2: no evidence of significant associations
Simpson et al. (2000)	Melbourne 1991–1996	Time-series	Percentage increases in all-ages group in warm months (November to March): Bsp 24-h avg, lag0: 5.5 (95% CI: 0.7, 10.5) for all-cause mortality and 18.0 (95% CI: 0.1, 39.0) for respiratory mortality per $10^{-4} m^{-1}$ NO_2 24-h avg, lag1: 0.4 (95% CI: 0.1, 0.6) for all-cause mortality and lag0: 1.5 (95% CI: 0.7, 2.2) for respiratory mortality per 1 ppb $PM_{2.5}$ 24-h avg, lag0: 0.4 (95% CI: 0.1, 0.7) for all-cause mortality and 1.2 (95% CI: 0.1, 2.3) for respiratory mortality per 1 $\mu g/m^3$ PM_{10} 24-h avg, lag0: 0.2 (95% CI: 0.0, 0.3) for all-cause mortality and 0.59 (95% CI: 0.0, 1.1) for respiratory mortality per 1 $\mu g/m^3$ O_3 max 1-h, lag02: 0.2 (95% CI: 0.1, 0.3) for all-cause mortality and O_3 max 4-h, lag0: 0.4 (95% CI: 0.1, 0.6) for respiratory mortality per 1 $\mu g/m^3$ No significant associations found for cardiovascular mortality in all of these air pollutants

Reference	Study location(s) and period	Method	Main findings*
Morgan et al. (1998b)	Sydney 1990–1994	Time-series	Percentage increases in all-ages group in all months (from 10^{th} to 90^{th} percentiles): Bsp avg 24-h, lag01: 2.6 (95% CI: 0.9, 4.4) for all-cause mortality, and 2.7 (95% CI: 0.3, 5.2) for cardiovascular mortality Bsp max 1-h, lag01: 2.5 (95% CI: 0.9, 4.2) for all-cause mortality and lag0: 3.0 (95% CI: 0.8, 5.1) for cardiovascular mortality NO_2 24-h avg, lag01: 2.7 (95% CI: 0.0, 5.3) for all-cause mortality O_3 max 1-h, lag0: 2.0 (95% CI: 0.4, 3.7) for all-cause mortality No significant association for respiratory mortality in any of these air pollutants
Simpson et al. (2005b)	Four cities (Brisbane, Melbourne, Perth and Sydney) 1996–1999	Multi-site time-series	Pooled relative risk estimates in all-ages group in all months: Bsp 24-h avg, lag1: 1.036 (95% CI: 1.012, 1.060) for all-cause mortality, 1.095 (95% CI: 1.017, 1.178) for respiratory mortality and 1.044 (95% CI: 1.009, 1.080) for cardiovascular mortality per $10^{-4}m^{-1}$ NO_2: max 1-h, lag1: 1.001 (95% CI: 1.001, 1.002) for all-cause mortality, lag1: 1.004 (95% CI: 1.002, 1.006) for respiratory and lag3: 1.002 (95% CI: 1.001, 1.003) for cardiovascular mortality per 1 ppb O_3 max 1-h, lag01: 1.002 (95% CI: 1.000, 1.004) for respiratory mortality per 1 ppb O_3 max 4-h, lag01: 1.003 (95% CI: 1.000, 1.005) for respiratory mortality per 1 ppb

Reference	Study location(s) and period	Method	Main findings*
EPHC (2010)	Seven cities (Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth and Sydney) 1998–2001	Multi-site case-crossover	Pooled percentage increases in all-ages group in all months (except for O_3 which are the results in warm months—November to April): CO max 8-h, lag01: no significant associations NO_2 max 1-h, lag01: 0.2 (95% CI: 0.0, 0.3) for all-cause mortality, 0.2 (95% CI: 0.0, 0.3) for cardiovascular and 0.4 (95% CI: 0.1, 0.7) for respiratory mortality per 1 ppb $PM_{2.5}$ 24-h avg, lag01: 0.4 (95% CI: 0.2, 0.6) for cardiovascular mortality per 1 $\mu g/m^3$ PM_{10} 24-h avg, lag01: 0.2 (95% CI: -0.0, 0.4) for all-cause mortality, and 0.2 (95% CI: 0.0, 0.3) for cardiovascular mortality per 1 $\mu g/m^3$ O_3 max 8-h, lag01: 0.1(95% CI: 0.0, 0.2) for all-cause mortality and 0.2 (95% CI: 0.0, 0.4) cardiovascular mortality per 1 ppb

Note: *Lags following with single numbers are single-day lags of the pollutant concentrations on the day indicated; for example lag0 is for the current day and lag1 is for the previous day. Lags following with two numbers are multi-day lags, which are running average concentrations for the number of days indicated; for example, lag01 is the running average of the current and previous days' concentrations. When multiple lags are examined for a given health outcome in these studies, only a lag that demonstrates the strongest association is presented in this table.

Table 2.2 Studies in Australia on morbidity associated with air pollution

Reference	Study location(s) and period	Health outcome	Method	Main findings*
Hansen et al. (2012)	Adelaide 2001–2007	Hospital admissions	Time-stratified case-crossover	Percentage increases in all-ages group (per 10 $\mu g/m^3$): $PM_{2.5}$ 24-h avg, lag01: 0.8 (95% CI: 0.2, 1.5) for total admissions and 2.7 (95% CI: 0.2, 5.3) for cardiovascular admissions in all months. The associations with cardiovascular admissions were stronger in the cool seasons. PM_{10} 24-h avg, lag01: 0.4 (95% CI: 0.2, 0.6) for total admissions in all months. No significant associations for respiratory admissions
Petroeschevsky et al. (2001)	Brisbane 1987–1994	Hospital admissions	Time-series	Relative risk estimates in all months: Bsp 24-h avg, lag04: 1.015 (95% CI: 1.006, 1.023) per $10^{-5} m^{-1}$ in all-ages group for total respiratory admissions NO_2: no significant associations in all months O_3 max 8-h, lag2: 1.023 (95% CI: 1.003, 1.043) per 1 pphm in all-ages group for total respiratory admissions SO_2: although several significant associations for respiratory and cardiovascular admissions were found, control diagnosis of digestive disorders was also positive.
Chen et al. (2007b)	Brisbane 1998–2001	Respiratory admissions	Time-series	4.0% increase in PM_{10} (24-h avg, lag0) (95% CI: 1.1, 6.9) in all-ages group in all months per 10 $\mu g/m^3$

Reference	Study location(s) and period	Health outcome	Method	Main findings*
EPA Victoria (2001)	Melbourne 1994–1997	Hospital admissions	Time-series	Relative risk estimates in all months: Bsp 24-h avg, lag02: 1.078 (95% CI: 1.012, 1.149) for respiratory 15–64 years, max 1-h, lag0: 1.148 (95% CI: 1.063, 1.240) for asthma 0–14 years, 24-h avg, lag1: 1.056 (95% CI: 1.021, 1.092) for cardiovascular 65+ years, and 24-h avg, lag0: 1.063 (95% CI: 1.019, 1.109) for IHD all ages per $10^{-4} m^{-1}$ CO max 8-h, lag02: 1.033 (95% CI: 1.010, 1.056) for total respiratory admissions 15–64 years, lag02: 1.033 (95% CI: 1.019, 1.048) for cardiovascular 65+ years lag04: 1.064 (95% CI: 1.036, 1.092) for asthma all-ages, and lag02: 1.037 (95% CI: 1.018, 1.056) for IHD per 1 ppm NO_2 24-h avg, lag04: 1.011 (95% CI: 1.007, 1.015) for respiratory 65+ years; lag04: 1.015 (95% CI: 1.010, 1.019) for asthma all ages, lag02: 1.005 (95% CI: 1.002, 1.007) for cardiovascular 65+ years, and lag0: 1.004 (95% CI: 1.001, 1.006) for IHD all-ages per 1 ppb O_3 max 4-h, lag2: 1.002 (95% CI: 1.000, 1.003) for respiratory admission 65+ years and 1.001 (95% CI: 1.000, 1.002) all-ages per 1 ppb. Strongest associations for O_3 found in the warm seasons, but for the other pollutants in the cool seasons
Erbas and Hyndman (2005)	Melbourne 1986–1992	COPD and asthma admissions	Time-series	Relative risk estimates (from 10^{th} to 90^{th} percentiles): NO_2 max 1-h, lag0: 1.060 (95% CI: 1.010, 1.110) for COPD and 1.050 (95% CI: 1.010, 1.090) for asthma (based on generalised additive model [GAM] approach). Associations for O_3 and air particle index and the admissions were sensitive to model specification

Reference	Study location(s) and period	Health outcome	Method	Main findings*
Erbas et al. (2005)	Melbourne 2000–2001	Childhood asthma ED visits	Time-series	Relative risk estimates (from 10th to 90th percentiles) in all months: NO_2 max 1-h, lag2: 1.150 (95% CI: 1.030, 1.270) in Western district and lag0: 0.830 (95% CI: 0.680, 0.980) in Inner Melbourne PM_{10} max 1-h, lag0: 1.170 (95% CI: 1.050, 1.310) in Inner Melbourne and lag0: 1.090 (95% CI: 1.010, 1.180) in Eastern district O_3 max 1-h: no significant linear associations but found significant non-linear associations at all levels of lag0 in Western and South/South-Eastern districts
Hinwood et al. (2006)	Perth 1992–1998	Hospital admissions	Time-stratified case-crossover	Percentage increases in: CO max 8-h: no significant associations for respiratory admissions, significant associations for cardiovascular admissions all-ages and 65+ years (lag1) NO_2 24-h avg, lag1: 0.6 for respiratory all-ages, lag1: 0.4 all-ages and lag2: 0.4 65+ years for cardiovascular admissions per 1 ppb $PM_{2.5}$ 24-h avg, lag2: 0.2 for respiratory admissions, lag3: 0.5 for pneumonia and lag2: 0.3 for asthma all-ages per 1 $\mu g/m^3$ O_3 max1-h and 8-h: no significant associations
Pereira et al. (2010)	Perth (south-west) 2002–2006	Childhood asthma ED visits	Time-stratified case-crossover	Odds ratios (per interquartile range): CO 24-h avg, lag1: 1.400 (95% CI: 1.060, 1.840) aged 0–4 years NO_2 24-h avg, lag1: 1.700 (95% CI: 1.080, 2.690) aged 0–4 years PM_{10} and O_3: no significant associations

Reference	Study location(s) and period	Health outcome	Method	Main findings*
Morgan et al. (1998a)	Sydney 1990–1994	Hospital admissions	Time-series	Percentage increases (from per 10^{th} to 90^{th} percentiles): NO_2 max 1-h, lag0: 5.3 (95% CI: 1.1, 9.7) in childhood asthma and 6.7 (95% CI: 4.3, 9.2) in cardiovascular 65+ years O_3 max 1-h: no significant associations PM 24-h avg, lag0: 2.8 (95% CI: 0.9, 4.8) and PM max 1-h, lag0: 2.7 (95% CI: 0.8, 4.7) in cardiovascular 65+ years
Jalaludin et al. (2006)	Sydney 1997–2001	Cardiovascular ED visits	Time-series	Significant associations with CO max 8-h, NO_2 max 1-h, PM 24-h avg (Bsp, $PM_{2.5}$ and PM_{10}), and SO_2 24-h avg for total cardiovascular disease, cardiac disease and IHD in the elderly 65+ No significant associations with O_3 Estimated relative risks were greater in cool than warm periods
Jalaludin et al. (2008)	Sydney 1997–2001	Childhood asthma ED visits	Time-stratified case-crossover	Significant associations with CO 8-h avg, NO_2 1-h avg, O_3 1-h avg, PM 24-h avg ($PM_{2.5}$ and PM_{10}), and SO_2 24-h avg in 1–4 years, with CO and PM in 5–9 years, and with CO and $PM_{2.5}$ in 10–14 years Estimated relative risks were greater in warm than cool months for NO_2, O_3, and PM

Reference	Study location(s) and period	Health outcome	Method	Main findings*
Simpson et al. (2005a)	Four cities (Brisbane, Melbourne, Perth and Sydney) 1996–1999	Cardiovascular and respiratory hospital admissions	Multi-site time-series	Pooled relative risk estimates in all-ages group For total cardiovascular admissions: Bsp 24-h avg, lag01: 1.086 (95% CI: 1.060, 1.112) per 10^{-4} m^{-1} NO_2 max 1-h, lag01: 1.002 (95% CI: 1.002, 1.003) per 1 ppb O_3: no significant associations Pooled relative risk estimates in the elderly 65+ years For respiratory admissions: Bsp 24-h avg, lag01: 1.055 (95% CI: 1.008, 1.105) for total respiratory, lag3: 1.071 (95% CI: 1.018, 1.128) for asthma and COPD, and lag01: 1.077 (95% CI: 1.005, 1.154) for pneumonia and acute bronchitis per 10^{-4} m^{-1} NO_2 max 1-h, lag01: 1.003 (95% CI: 1.002, 1.004) for total respiratory and 1.003 (95% CI: 1.001, 1.005) for pneumonia and acute bronchitis per 1 ppb O_3 max 4-h, lag3: 1.001 (95% CI: 1.000, 1.003) for asthma and COPD per 1 ppb

Reference	Study location(s) and period	Health outcome	Method	Main findings*
EPHC (2010)	Seven cities (Auckland, Brisbane, Canberra, Christchurch, Melbourne, Perth and Sydney) 1998–2001	Hospital admissions	Multi-site case-crossover	Pooled percentage increases in the elderly 65+ years for total cardiovascular admissions: CO max 8-h, lag01: 2.5 (95% CI: 1.0, 4.0) per 1 ppb NO_2 max 1-h, lag01: 0.3 (95% CI: 0.2, 0.4) per 1 ppb $PM_{2.5}$ 24-h avg, lag01: 0.3 (95% CI: 0.1, 0.5) per 1 µg/m^3 PM_{10} 24-h avg, lag01: no significant associations O_3 max 8-h, lag01: no significant associations Pooled percentage increases in children for total respiratory admissions: CO max 8-h, lag01: no significant associations NO_2 max 1-h, lag01: 0.3 (95% CI: 0.1, 0.5) for 1–4 age group and 0.5 (95% CI: 0.2, 0.9) for 5–14 age group per 1 ppb $PM_{2.5}$ 24-h avg, lag01: 0.6 (95% CI: 0.3, 1.0) for <1 age group and 0.4 (95% CI: 0.2, 0.7) for 1–4 age group per 1 µg/m^3 PM_{10} 24-h avg, lag01: 0.2 (95% CI: 0.0, 0.4) for <1 age group; 0.2 (95% CI: 0.1, 0.4) for 1–4 age group and 0.3 (95% CI: 0.0, 0.5) for 5–14 age group per 1 µg/m^3 O_3 max 8-h, lag01: 0.2 (95% CI: -0.0,0.5) for 1–4 age group per 1 ppb

Note: *Lags following with single numbers are single-day lags of the pollutant concentrations on the day indicated; for example lag0 is for the current day and lag1 is for the previous day. Lags following with two numbers are multi-day lags, which are running average concentrations for the number of days indicated; for example, lag01 is the running average of the current and previous days' concentrations. When multiple lags are examined for a given health outcome in these studies, only a lag that demonstrates the strongest association is presented in this table.

Although less attention has been paid to the health effect associated with exposure to CO due to its low ambient levels, some studies have found that there is a significant association between CO concentration and morbidity (Environmental Protection and Heritage Council 2010; EPA Victoria 2000; Hinwood et al. 2006; Pereira et al. 2010; Jalaludin et al. 2006; Jalaludin et al. 2008). For most of the cities studied to date, NO_2 has been associated with both cardiovascular and respiratory illnesses, including within the results of the two multi-site studies. Although there are no associations found between NO_2 and hospital admissions in Brisbane overall, when stratified by season, there was a significant positive association in warm seasons only (Petroeschevsky et al. 2001).

Significant associations between concentrations of PM (Bsp, PM_{10} and $PM_{2.5}$) and both cardiovascular and respiratory morbidity have been evident, with only a few exceptions. For example, there was no evidence of associations between PM_{10} and respiratory hospital admissions and asthma ED visits in Adelaide (Hansen et al. 2012) and Perth (Pereira et al. 2010) respectively. For O_3, there is agreement across all the studies indicating strong associations between concentrations of this pollutant and respiratory morbidity, particularly asthma. However, these studies found only weak associations between O_3 and cardiovascular disease, in particular when O_3 measurements are considered for the whole year. Notably though, a study in Melbourne showed a significant association between O_3 and cardiovascular admissions in the elderly aged above 65 years in warm months (EPA Victoria 2001).

Results from heterogeneity tests in the two multi-site studies lead to slightly different conclusions in relation to the association between air pollution and morbidity. In the SPIRT study, hospital admissions were divided into two main categories: cardiovascular and respiratory disease. The cardiovascular category was divided further into all cardiac and IHD subgroups, while the respiratory category had four subgroups, including total respiratory, asthma, COPD and pneumonia-acute bronchitis. The pollutants of interest included Bsp, NO_2 and O_3. That study reported strong differences between the four cities in the pooled estimates for total respiratory admissions attributable to NO_2 and Bsp. For cardiac admissions, although significant heterogeneity was found for NO_2 and Bsp in some lagged-days exposures, an overall conclusion was that the pooled estimates were applicable for all cities. In the EPHC study, it was concluded that there was no evidence to suggest heterogeneity of most pooled estimates for different categories of cardiovascular admissions, including total cardiovascular, all cardiac, IHD, stroke, myocardial infarction,

cardiac failure and arrhythmia with the pollutants investigated (Bsp, CO, NO_2, PM_{10}, $PM_{2.5}$ and O_3). One exception is for the combined estimates associated with CO for cardiac and total cardiovascular hospital admissions for the elderly aged above 65 years. For the respiratory admissions of interest, which included four subgroups namely total respiratory, asthma, COPD and pneumonia and acute bronchitis, except for the pooled estimate for the association of NO_2 with asthma in the 5–14 years group, testing for heterogeneity revealed no evidence of differences between cities for combined risk estimates associated with all air pollutants examined and all respiratory admissions categories of interest. This leads to a conclusion that the pooled estimates can be applied across all of the cities used in these analyses.

2.2.2.5 The health effect of air pollution in different age groups based on Australian studies

Age has been commonly identified as an important effect modifier for the associations between air pollution and health outcomes in most Australian studies. In general, associations for different categories of cardiovascular disease are larger and more significant in the elderly. For example, Simpson et al. (1997) found that significant associations between cardiovascular mortality and Bsp and O_3 were apparent only in the elderly aged above 65 years in Brisbane. Results from a study in Melbourne showed that associations between concentrations of Bsp, CO and NO_2 and cardiovascular admissions were stronger in the elderly aged above 65 years, compared with other age groups (EPA Victoria 2001).

In terms of the effects of air pollution on asthma, results from most studies suggest that children aged below 15 years are the most vulnerable population. Nevertheless, some studies in Australia have also found significant associations in other age groups. For example, Hinwood et al. (2006) found strong associations between $PM_{2.5}$ and asthma admissions in the all-ages group, as well as for children aged below15 years in Perth. Similarly an average estimate for the association between $PM_{2.5}$ and asthma admissions based on combining results across the cities in the EPHC study is positively significant in the elderly aged above 65 years (Environmental Protection and Heritage Council 2010). In the SPIRT study, pooled estimates indicated significantly increased asthma admissions in relation to Bsp and O_3 concentrations in the elderly aged above 65 years (Simpson et al. 2005a).

2.2.2.6 Seasonal variations in the health effects of air pollution based on Australian studies

Seasonal variation in the health effects of air pollution has been analysed in some of the studies reviewed here. Australia can be divided into six main climatic zones (equatorial, tropical, subtropical, dessert, grassland and temperate) (Australian Bureau of Meteorology 2013). The temperate zone where five capital cities are situated (Adelaide, Canberra, Melbourne, Sydney and Hobart) can also be classified further into nine sub-zones. Cities included in the Australian studies lie within different climatic zones and sub-zones, and this may provide an explanation for the variable findings of seasonal modification of health impacts of air pollution that have been reported.

In Adelaide, where the climate is classified as temperate, with a warm summer and moderately dry winter, Hansen et al. (2012) found positive associations between PM—both $PM_{2.5}$ and PM_{10}—and total and cardiovascular admissions, which were mainly apparent and of greater magnitude in cool seasons. The author attributed the stronger effect in cool seasons to two factors: synoptic weather patterns and PM components. Although it was not discussed in that paper, as suggested by a study of source apportionment in Australian cities, including Adelaide (Chan et al. 2008), the stronger effect of PM in cool seasons may be due to higher levels of $PM_{2.5}$ (indicated by high $PM_{2.5}/PM_{10}$ ratio), which are more toxic than PM_{10} (Heal et al. 2012).

In Sydney, where the climate is also temperate but with a hot summer and no dry season, Jalaludin et al. (2006) found greater cardiovascular risks in the elderly in cool months than warm months for all pollutants examined (CO, NO_2, PM and SO_2) except O_3. In the same setting of Sydney but in different age groups, another study found greater effects of NO_2, PM and O_3 on childhood asthma ED visits in warmer periods (Jalaludin et al. 2008). The mixed findings on the seasonal impact of NO_2 and PM on health outcomes in these two studies in Sydney may be the result of an interaction of multiple effect modifiers, in this case at least, season, age and disease.

Apparently, contradictory results for the modifying effect of season on health effects of PM and NO_2 were also found in two studies in Melbourne (Simpson et al. 2000; EPA Victoria 2001). Simpson et al. (2000) found mortality risks attributed to Bsp and NO_2 stronger across different age groups in warm months. On the contrary, EPA Victoria (2001) estimated that risks for cardiovascular and respiratory admissions associated with

the same pollutans were greater in winter months. However, these two studies considered different time windows and health outcomes of interest (mortality vs. morbidity), so that it is difficult to properly evaluate these contradictory results regarding seasonality. One agreed conclusion from these two studies is that the impact of seasonality in Melbourne for the effect of O_3 on health follows a typical pattern in temperate locations where a stronger effect is found in warm periods.

In Brisbane, where the climatic conditions are subtropical and do not fluctuate as much as the other temperate cities in Australia, the effect modification by season may not be strongly manifested. Petroeschevsky (2001) found no substantial seasonal differences in associations between Bsp, NO_2 and O_3 and cardiovascular and respiratory hospital admissions in Brisbane. The authors pointed to the influence of the subtropical climate—wet summers and high sunshine winters—in Brisbane so that seasonal variation is not as strong as in temperate cities. Similar findings on the weak seasonal effect were also found in analyses based on data collected in Brisbane during 1998 to 2001 as part of the EPHC study.

In the EPHC study, seasonal variation is distinct, particularly in Perth and Melboune. The associations between NO_2 and PM concentrations and most of the health outcomes examined (e.g., NO_2 in relation to cardiovascular and respiratory mortality for all ages, $PM_{2.5}$ in relation to respiratory mortality aged above 75 years and total respiratory admissions for all ages) were postive and greater in the winter period (Environmental Protection and Heritage Council 2010). In contrast, postive associations between O_3 and health outcomes examined were usually found only in the warm period.

2.3 Interaction between temperature and air pollution on the health effects of air pollution

There is a large body of evidence, as large as the evidence of the adverse effects of air pollution on health, showing the effects of temperature on health (e.g., Baccini et al. 2008; Turner et al. 2012; Yu et al. 2012; Stafoggia et al. 2008). There is also a wealth of evidence showing the influence of temperature on the levels of air pollution. Therefore, a standard approach in time-series studies on air pollution and health is to include temperature as a confounder (Buckley et al. 2014). Driven by concern for the warmer climate trends over the recent decades and maturing knowledge about temperature-related health effects, investigating interaction effects of simultaneous exposure to air pollution and temperature

on health has gained increased attention from the scientific community. These studies have applied the conventional methods commonly used in epidemiological time-series or case-crossover studies of air pollution with some modification to capture modifying effects of temperature on the health effects of air pollution, and vice versa.

This section contains a review of studies, from both overseas and in Australia, that have investigated potential interaction effects of temperature and air pollution on human health. Findings from these studies are first discussed, and then followed by a summary of the study designs and statistical methods used in these studies. Plausible mechanisms are also reviewed and discussed at the end of this section to understand results from these studies.

2.3.1 Evidence from epidemiological studies

In general, investigating the effects of interactions between air pollution and temperature on health can be undertaken under two different circumstances. Most commonly, interaction effects are explored under day-to-day variation of weather and air pollution. Alternatively, relationships can be examined during extreme temperature episodes such as heatwaves. The latter approach is useful to help gain more understanding and reinforce the evidence of the interaction effects investigated under more usual day-to-day circumstances.

2.3.1.1 Studies overseas investigating temperature modifying PM- and O_3-related health effects under day-to-day circumstances

Under day-to-day day circumstances, O_3 and PM have been the main pollutants investigated to examine any interactions with temperature that modify their effects on health. Among studies that have investigated the interaction between temprature and PM on health, different magnitudes and patterns of the interaction have been found (Roberts 2004; Stafoggia et al. 2008; Qian et al. 2008; Li et al. 2011; Carder et al. 2008).

Through analysing data from two counties in the US, Roberts (2004) observed an interaction between temperature and PM_{10} in which associations between PM_{10} and all-cause mortality in the elderly were found only on days that daily mean temperatures were above the 10^{th} percentile. Similarly, findings from a study in nine Italian cities suggested an interaction of PM_{10} and apparent temperature on mortality, whereby the PM_{10} effects were higher on warmer days (temperatures were greater than the 50^{th} percentile; approximately 14.5°C averaged across the cities) (Stafoggia et al. 2008). Two studies in China have also provided evidence to support an interaction effect in that stronger associations of PM_{10} on various mortality outcomes were identified on high temperature days (Qian et al. 2008; Li et al. 2011). In contrast to the aforementioned studies, Carder et al. (2008) found evidence of an interaction effect between black smoke and temperatures on respiratory mortality based on the data from three Scottish cities, where the black smoke effect was apparent only at extremely low temperatures (lower than 11°C).

Surprisingly, despite the frequent concurrence of high levels of O_3 on warm days in polluted areas, fewer studies—compared with PM—have specifically investigated the modification effect of temperature on the health effects of O_3 (Ren et al. 2008a; Pattenden et al. 2010; Ren et al. 2009; Ren et al. 2008b). The lesser attention paid to O_3 may be due to the fact that high levels of PM occur throughout the year. This allows studying the interaction effects between PM and temperature at both lower and upper extreme temperatures, and not necessarily limited to warm seasons as occurs for O_3. Further, as estimated by the recent Global Burden of Disease study (Lim et al. 2012), premature deaths associated with ambient PM are generally greater by a factor of 20, compared to those associated with ambient O_3.

Findings from two studies that used the NMMAPS data from 1987 to 2000 to combine health risk estimates across multiple cities indicated interaction effects between O_3 and

temperature, which showed stronger associations between O_3 and total and cardiovascular mortality at higher temperature levels (Ren et al. 2008a, 2009). However, the interaction effects were heterogeneous between the cities examined; local climate conditions may have contributed to the variation in the effects. Based on these two studies, regions where O_3 and temperature were highly correlated—particularly in the northern and north-eastern parts of the US—tended to have more homogeneous and stronger interaction effects. In addition to temperature being a modifier of the relationship between O_3 and health outcomes, one study has shown that O_3 may modify the relationship between temperature and cardiovascular mortality (Ren et al. 2008b).

A study conducted in the UK found evidence of the interaction effect of temperature and O_3 on different causes of mortality. Here the effect appeared to be strongest in London, and was generally weaker or not noticeable in 14 other conurbations concurrently examined (Pattenden et al. 2010). In London, an additional relative risk of the O_3 effects on hot days (2-day averages of mean temperature were above the 95th percentile, 19.9°C) was 1.009 (95% CI: 1.003, 1.015) per 10 $\mu g/m^3$ increase of O_3. Despite the sparse interaction effects seen in other conurbations, a pooled relative risk of the O_3 effects across the conurbations suggested that there was a greater health risk on hot days compared to cooler days—with no evidence of heterogeneity across regions.

In addition to the studies aforementioned, some studies have considered PM and O_3 together and tested how temperature interacts with them to cause adverse effects on health. Ren et al. (2011) assessed associations between apparent temperature and heart rate variability in an ageing male population in Boston, Massachusetts. They observed that the strongest associations were on summer days when O_3 was higher than its median concentration (22.3 ppb). In that study, exposure to ambient $PM_{2.5}$ concentrations in the summers did not modify the temperature effects on heart rate variability.

Another study, which investigated the temperature modification of the effect of air pollutants (NO_2, O_3, PM_{10} and SO_2) on a range of mortality outcomes in Shanghai, China, found that only the PM and O_3 effects were modified at extreme lower ends of temperature (Chen and Kan 2012). Most of the risk estimates of O_3 at low temperature levels at different temperature cut-points tested (5th, 10th, 15th, 20th and 25th percentiles) were significant and three to four times greater than the relative risks estimated at medium and high temperature levels. For PM_{10}, although the magnitudes of the risk estimates at low

temperature levels were relatively close to those for other temperature levels, the risk estimates were significant only at low temperature levels (below the 15th percentile). The stronger association of the PM effect on cold days was explained by two key factors: larger death counts and higher PM concentrations. In contrast to other studies that have reported stronger adverse effects of O_3 on health outcomes on days with high temperatures, the authors of that study speculated that having no evidence of stronger O_3 effects on hot days might be related to the hot and humid weather conditions in Shanghai in summer. Such climate conditions could induce people to be less exposed to air pollution as they spent more time indoors and used air conditioning more often—which could, to a great extent, prevent penetration of O_3 into the building.

2.3.1.2 Studies overseas investigating temperature modifying health effects related to other air pollutants under day-to-day circumstances

Other pollutants including CO, NO_2 and, to a lesser extent, SO_2, have also been investigated for their interaction effects with temperature. Lin and Liao (2009) examined combined effects of temperature and various pollutants (CO, NO_2, PM_{10} and SO_2) on all-cause and cardiovascular mortality in Kaohsiuang, Taiwan. Among the pollutants examined, only an interaction effect of temperature and CO was found. The associations between lag 2-day CO and cardiovascular mortality were greater and significant only at lower temperature quartiles (7% increase in cardiovascular mortality per 0.2 ppm increase of CO at the 1st temperature quartile; 19.7°C). In contrast, risk estimates of current day CO exposure on all-cause mortality were larger and significant at higher temperature quartiles (4.3% and 5.1% increases in all-cause mortality per 0.2 ppm increase of CO at the 2nd temperature quartile [24.8°C] and at the 3rd temperature quartile [27.6°C], respectively). The authors applied different hypotheses to explain these contradictory results. More prominent associations between cardiovascular illnesses and deaths in cool temperatures were used to explain the antagonistic effects of CO and cool temperatures on cardiovascular mortality. The physiological stress of heat exposure and greater chances of exposure to the pollutant via different pathways on warmer days were postulated as the key underlying mechanism of the synergistic effect of temperature and CO on all-cause mortality.

Another study in Seoul Korea explored the interaction effects of a group of air pollutants (CO, NO_2, O_3, PM_{10} and SO_2) and temperature on mortality outcomes (Park et al. 2011).

Among the pollutants studied, there were interaction effects of temperature on mortality only in relation to SO_2 concentration. The risk estimate of SO_2 on non-accidental mortality was greater on hot days (> 26.2°C) by a factor of four, compared with the overall relative risk estimated for the entire temperature range. For PM_{10}, the association with cardiovascular mortality was generally greater when temperatures were warmer, but the results indicated that the synergistic effects were not as distinct as for SO_2. The authors linked the increases of the PM_{10} effect in association with temperature to possible rapid formation of sulphate components at high temperatures. The diminution of the PM_{10} effect in the two-pollutant model adjusted for SO_2 appeared to support this explanation.

2.3.1.3 Australian studies investigating temperature modifying air pollution-related health effects under day-to-day circumstances

In Australia, a few studies have been conducted to explore the interaction effect of temperature and air pollution on a range of health endpoints. In Brisbane, where the climate is tropical and simultaneous exposures to air pollution and heat provide a plausible climate change scenario, two studies were conducted to investigate the interaction effects (Ren et al. 2006; Ren and Tong 2006). The first of these two studies explored how PM_{10} modified the association between temperature and cardiorespiratory diseases (Ren et al. 2006), whereas the other considered the interaction effects in reverse to the first study (Ren and Tong 2006). The authors found effect modification in both studies in that PM_{10} and temperature acted synergistically, resulting in stronger impacts on most of the health outcomes examined at different lag days tested (lags of 0, 1 and 2 days).

Hu et al. (2008) explored the interaction effect of air pollution and temperature on all-cause mortality in Sydney. Among the air pollutants studied (CO, NO_2, O_3, PM_{10} and SO_2), only SO_2 was significantly associated with mortality. Based on a time-series classification and regression tree (CART) model, exposure to SO_2 at levels above 0.315 pphm at temperatures ranging from 29–32°C resulted in an increase in mortality by 12.1%, considerably higher than an increase in all-cause mortality by 7.3% purely from exposure to temperatures above 32°C.

2.3.1.4 Temperature modifying air pollution-related health effects under heatwaves

Since the late 20th century, the occurrence of heatwaves and the number of hot days has noticeably increased. Not only can exposure to heat result in increases in adverse health

impacts during extreme temperature events, but high air pollution accompanying the heat may also play a significant role. Consideration of air pollution during heatwave episodes to probe into simultaneous health effects of high temperature and air pollutants has been undertaken by a few studies.

In the Northern Hemisphere, the most severe heatwave affecting many European countries was witnessed in 2003. For the 2003 heatwave, two studies were undertaken to explore a contribution of O_3 and the potential interaction of temperature and O_3 on mortality in France (Dear et al. 2005; Filleul et al. 2006). Both studies found evidence of excess deaths caused by higher O_3 concentrations during the heatwave period. Filleul et al. (2006) reported a joint effect of temperature and O_3 with ratios of excess deaths due to O_3 relative to temperature ranging from 2.57% to 85.34% in nine cities during the period 3 to 17 August 2003. Dear et al.(2005) estimated attributable deaths of exposure to maximum temperature, minimum temperature and O_3 in their analysis of data for the heatwave period (25 June to 19 August 2003) in 12 cities, taking account of distributed lag effects. The results from that study suggested that the excess deaths due to exposure to O_3 ranged from -6 for Nice to 551 for Paris (calculated by subtracting an estimate based on the model accounting for the effects of exposure to all the three variables with an estimate based on the temperature model that accounted for the effects of exposure to maximum and minimum temperature only). Considering the total deaths in all the 12 cities, deaths related to O_3 exposure during the heatwave were approximately one-third of those related to temperature. In the Southern Hemisphere, Tong et al. (2010a) estimated that exposure to increased O_3 concentrations in the range of 6.0 to 19.6 ppb during the 2004 heatwave in Brisbane, Australia, contributed around one-fourth of excess non-external all-cause mortality; the remainder was related to heat exposure.

Based on the results from these three studies, it appears that exposure to high O_3 concentrations is a significant contributor to adverse health effects occurring during heatwaves. However, since such evidence has been drawn from only limited studies, further research on the role of air pollutants and their interactions with temperature on health during heatwave episodes is needed.

2.3.2 Methods in epidemiological studies examining the interaction effects of temperature and air pollution on health

Figure 2.1 illustrates key methods and their links that have been applied in previous studies to investigate the interaction effects of temperature and air pollution on health. The methods can be classified as: i) non-stratification and ii) stratification approaches.

The non-stratification approaches aim at initial exploration of the existence of the interaction effects. If an *a priori* assumption of linear relationships between air pollution and health is made, data are fitted with parametric regression models by including a linear interaction term of temperature and .air pollution. If non-linear relationships are assumed, data are fitted with either parametric or non-parametric regression models. For the parametric regression models, bivariate response functions have been commonly chosen from studies in the past to visually display the non-linear relationships of temperature and air pollution on health. For the non-parametric approaches, thin plate spline functions and CART have been chosen to identify the interaction effect.

Stratification approaches that use temperature cut-points to split temperatures into different strata have been separately applied or used in combination with the non-stratification approaches. The main goal of the stratification approaches, in addition to identifying the interaction effect, is to quantify relative risks that vary between temperature strata if the interaction effect is apparent. The choice of temperature cut-points can be arbitrary or guided by the relationship of temperature, air pollution and health identified from the non-stratification approaches. If an arbitrary choice of temperature cut-points is made, a sensitivity analysis is usually conducted to test the degree to which varying temperature cut-points would affect the magnitude of relative risk estimates and whether using other alternative cut-points would be capable of detecting the interaction effects.

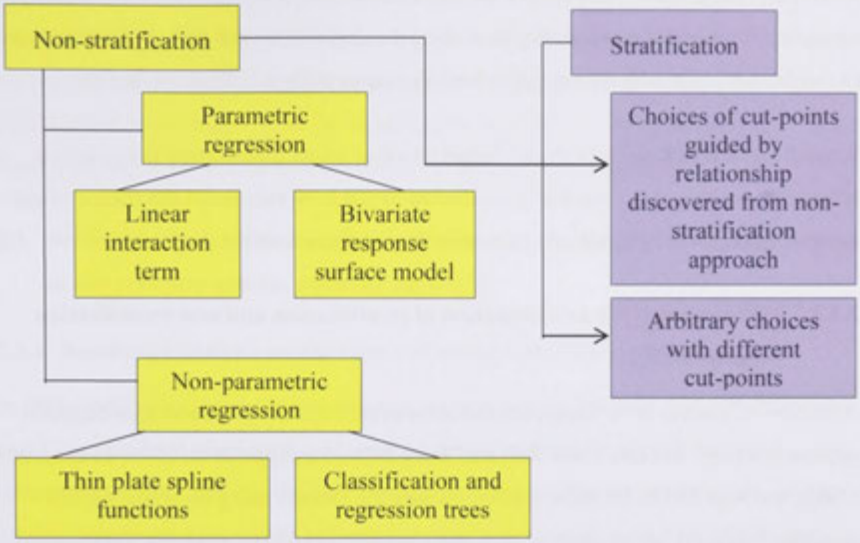


Figure 2.1 Key methods and their links for examining the interaction effect of temperature and air pollution on health outcomes

2.3.2.1 Studies applying a single approach

Most studies that used only a single method have favoured stratification approaches (Carder et al. 2008; Qian et al. 2008; Lin and Liao 2009; Park et al. 2011; Pattenden et al. 2010; Ren et al. 2011). There has been only one exception, in which Hu et al. (2008) determined an interaction of temperature and SO_2 on all-cause mortality by relying on a CART regression model.

In terms of stratification strategy, the number of strata for temperatures or air pollution concentrations can range from two to four. For studies in which cut-points were arbitrarily selected, decisions were made based on either central tendencies or a range of different percentiles of the modifier examined. For example, Carder et al. (2008) and Ren et al. (2011) divided temperatures into two strata by using mean and median respectively as a cut-point in their studies. Lin and Liao (2009) and Park et al. (2011) used temperature quartiles to examine differences of air pollution and health effects between the temperature levels. Qian et al. (2008) examined the modifying effects of temperature on the association between PM_{10} and mortality by using two extreme cut-points at the 5^{th} and 95^{th} percentiles. The study by Pattenden (2010) was the only one—among those solely dependent on the

stratification approach—that harnessed information from a companion study identifying a temperature threshold on mortality in making the decision on cut-points of hot days where O_3 might synergistically interact with heat and cause stronger effects on health.

Regardless of which strategy these studies chose to select temperature cut-points, a sensitivity analysis was commonly conducted to test how influential the choice of cut-points was on the magnitude and pattern of the interaction effect.

2.3.2.2 Studies applying a combination of stratification and non-stratification approaches

A number of studies have used a combination of non-stratification and stratification approaches (e.g., Roberts 2004; Ren and Tong 2006; Stafoggia et al. 2008; Li et al. 2011; Cheng and Kan 2012). Of these studies, parametric models using bivariate surface response functions, at the stage of non-stratification, have been primarily fitted aiming to observe the existence and patterns of the interaction effects.

Non-stratification parametric models, in which the association between air pollution and health effects is assumed to be linear and an interaction term of temperature and air pollution is added to the model, have been analysed in only two studies conducted by the same authors (Ren et al. 2006; Ren and Tong 2006). In these two studies, both parametric and non-parametric models were fitted in parallel. The use of non-parametric models allows more flexibility to capture relationships between temperature, air pollution and health effects in which nonlinearities among them might exist.

Although patterns of the interaction effects observed from the non-stratification models can be informative to decide what would be appropriate cut-points to be used at the stage of stratification, only a few studies have embraced such a strategy (Roberts 2004; Li et al. 2011). This might be due to inconsistent interaction patterns between various health outcomes and locations studied. Moreover, the interaction patterns observed could not provide a definite range of the effect modifier in guiding selection of its cut-points. For example, there was no sign of a threshold of temperature and PM_{10} on mortality in non-parametric models fitted for data from two Italian cities, and the interaction patterns observed in these two cities were different from each other (Stafoggia et al. 2008). Cheng and Kan (2012) found different patterns of the interaction between O_3 and temperature on cardiovascular and respiratory mortality in Shanghai. In that study, the stronger O_3 effect

on cardiovascular mortality was clearly observed when temperatures were above approximately 15°C. At above the same temperature threshold, however, risk estimates of respiratory mortality were relatively constant across the range of high O_3 concentrations. Therefore, when these studies used the stratification approaches, they commonly considered sensitivity of the interaction effects at alternative cut-points.

2.3.2.3 Sensitivity analyses undertaken in studies investigating the interaction effects of temperature and air pollution on health

2.3.2.3.1 Sensitivity analysis on the choice of temperature cut-points

When the stratification approach is conducted, irrespective of whether a non-stratification approach has also been employed, it is worthwhile to consider a range of alternative temperature cut-points. For the studies in which high ends of the temperature range were selected and alternative cut-points beyond that range were tested in sensitivity analyses, conclusions drawn on the interaction effects remain largely unchanged. For example, Qian et al. (2008) and Pattenden et al. (2010) found that estimated PM_{10} and O_3 effects on mortality were similar when alternative cut-points higher than the 95th percentile—the primary cut-point selected for the high temperature stratum—were used. In the studies reported by Roberts (2004) and Cheng and Kan (2012), a combination of the 5th and 95th percentiles and the 15th and 85th percentiles respectively were used to be primary cut-points. They also reported a relatively wide range of cut-points, 2.5th to 15th percentiles and 5th to 25th percentiles respectively, in which the health risks estimated did not substantially change.

In studies in which the initial cut-points chosen were based on central tendencies or quartiles, the health effects estimated did appear to be sensitive to the alternative cut-points when these were below the extreme ranges. Park et al. (2011) observed greater mortality risks of SO_2 at the 80th, 85th, 90th, 93rd and 95th percentiles of temperature cut-points compared with the risk estimated by using the 75th percentile as the primary cut-point for the high temperature stratum. Stafoggia et al. (2008) found that interaction terms of high temperature strata were not statistically significant when warmer cut-points (each percentile between the 76th to 90th percentiles) were used as alternatives instead of the main cut-point at the 75th percentile. In addition, the significant estimates of PM_{10} effects in the highest temperature stratum were gradually diminished towards zero for the greater alternative cut-points above the 75th percentiles.

2.3.2.3.2 Sensitivity analysis on the choice of lags

It is well established that an adverse health effect due to exposure to some environmental factors, including air pollution and temperature, can extend over a certain period after the time of exposure (Samoli et al. 2009; Muggeo 2007; Schwartz 2000; Anderson and Bell 2009). This delayed effect is generally referred to as a lag effect. A number of studies investigating interaction effects have, to a certain extent, attempted to address the issue of lag effects (e.g. Roberts 2004; Ren and Tong 2006; Carder et al. 2008). These studies mainly assumed cumulative effects induced by exposure to temperature and air pollution over a short period (less than a week). Of these studies, some adhered to a multi-day moving average of temperature and air pollution when they explored the interaction effects and quantified the health risks (Qian et al. 2008; Stafoggia et al. 2008; Park et al. 2011). This is a reasonable approach as it has been shown in previous studies that using a moving average—a special case of unconstrained distributed lag models with an equal weight given to each day of the effect over the lagged period—can result in a more accurate estimate of health risks than considering only a single-day lag (Schwartz 2000). The use of an average of lags 0 and 1 applied consistently for both air pollution and temperature has been more common than other lag averages (Pattenden et al. 2010; Park et al. 2011; Stafoggia et al. 2008; Qian et al. 2008).

An alternative to handle the delayed effects is to use multiple lags—single-day lags or multi-day averages—to demonstrate the extent to which varying lags would impact on the health effect estimates. To simplify this, although the lagged effects should be handled for both temperature and air pollution, all the studies exploring interactions varied only lags of the environmental factor of interest and fixed lags of the effect modifier. Making a comparison between multiple lags was undertaken in a few studies at the stage of stratifying the effect modifier into different strata (Roberts 2004; Ren and Tong 2006; Carder et al. 2008). Results from these studies were suggestive of distributed lag effects in which the highest risk estimate was usually found at lag 0 and gradually reduced over a few days later. Compared with using a single-day lag, multi-day moving averages give larger health effect estimates (Roberts 2004; Pattenden et al. 2010; Carder et al. 2008).

Among the studies investigating the interaction effects, only Carder et al. (2008) considered the lagged effects of temperature and black smoke over a period longer than one week. The authors found that the black smoke effects on all-cause, respiratory and

non-cardiorespiratory mortality at a moving average of lags 13–18 were significant and largest or approximately equal to short-term lags (lag 0 or a moving average of lags 1–6). This highlights the importance of considering delayed effects that might be possibly prolonged over a period longer than a week.

2.3.2.4 Other aspects of model specification

Some aspects of model specification, for example, degrees of freedom of terms applying to smoothers and the inclusion of other co-pollutants in the model, have also been considered in studies investigating interaction effects. Evidence from past studies suggests that varying these variables in a statistical model for studying adverse health effects of air pollution can influence estimates of the relative risks (Bateson et al. 2007; Wong et al. 2008).

Roberts (2004), Ren and Tong (2006), Qian et al. (2008), Lin and Liao (2009), Park et al. (2011) and Pattenden et al. (2010) tested the impacts on the health effect estimates of changing the degrees of freedom applied for smoothing functions for different variables including temperature, time and season. Mostly these studies found that the choice of degrees of freedom had a minor impact on the overall conclusions for the interaction effects and the health effect estimates as long as the changes were not substantial, for example, less than a factor of two from the original choice.

Qian et al. (2008), Park et al. (2011) and Li et al. (2011) included a second co-pollutant into their single-pollutant models to examine this variation on the model output. Inconsistent findings on the confounding effects of multiple pollutants were found from these studies. Park et al. (2011) found that the PM_{10} effects on non-accidental mortality at high temperatures were considerably diminished when SO_2 was included as a co-pollutant. In contrast, Qian et al. (2008) and Li et al. (2011) did not find substantial changes of estimates of the PM_{10} effects after introducing SO_2 into the model. In part, the mixed findings from these three studies may be explained by the different mixes of air pollutants and the diversity of climate patterns inherited in different locations. The climate patterns and the composition of the air pollution in the studies of Qian et al. (2008) and Li et al. (2011) were relatively similar as these two studies were both conducted in China and in cities—Wuhan and Tianjin—that are strongly influenced by the monsoon. This is in contrast to the settings in the study of Park et al. (2011) that was conducted in Seoul, Korea, a city with a temperate climate. One reflection based on these inconsistent findings

from the co-pollutant models is that it warrants further investigation of the interaction effects to consider inclusion of co-pollutants to obtain more accurate estimates.

2.3.3 Plausible mechanisms

Underlying mechanisms to explain the interaction effects between air pollution and temperature on health found in the epidemiological studies are mainly associated with three pathways. The first two pathways are about changes in doses and activity patterns of population exposure to temperature and air pollution, whereas the other pathway might be related to physiological responses in the human body when it is concurrently exposed to these two environmental factors.

In the first pathway, the doses or levels of ambient air pollution that a population is exposed to are strongly influenced by temperature profiles, occurring via a number of processes. Formation and chemical reactions of some air pollutants, for example, NO_x, O_3 and some components of PM, such as sulphate, vary across temperature ranges. The most distinct example is O_3, a pollutant that requires warm temperatures and sunlight for its formation (Mark Z. Jacobson 2002). High O_3 concentrations are frequently recorded on warm days in summer, in particular for subtropical cities. In addition to these chemical processes, atmospheric physical processes, such as inversion, turbulence and wind, are also important in determining air pollutant concentrations and these are temperature-related. Emissions of some pollutants are substantially dependent on temperature. For example, PM concentrations in temperate locations are high in winter when the use of wood heaters is high. At the upper temperature extremes, different emissions sources from biomass burning and forest fires—events that are prevalent on warm days in summer—can also cause high PM concentrations. By having high atmospheric pollution levels, in particular at extreme temperatures, people are likely to be exposed to higher doses of air pollution as a result.

Population responses to extreme temperatures have been hypothesised as a second factor related to the alteration of health risks of air pollution. Roberts (2004) and Stafoggia et al. (2008) reported that stronger PM effects on mortality were detected on hot days. The authors explained that this finding may be due to greater exposure to air pollution as people spend more time outdoors or have open windows, making measurements from fixed monitoring sites a better indicator of actual individual exposures. In addition, changes in behaviour in response to extreme temperatures could lead to exposure to higher or lower

doses of air pollution depending on climate conditions. Ren et al. (2008a) and Cheng and Kan (2012) explained that attenuated O_3-related health effects found in summers in tropical climate settings were possibly a consequence of less exposure to O_3 because people tended to have less outdoor activities. For populations living in tropical cities, reliance on air conditioning in summer can both cool down hot temperatures and decrease exposure to outdoor air pollution when people stay indoors, resulting in having lower risks to temperature and/or air pollution (Bell et al. 2009; Bell and Dominici 2008).

Patho-physiological changes in the body potentially induced by simultaneous exposure to extreme temperatures and air pollution have been most frequently used to explain underlying mechanisms of the interaction effects. The most well-known physiological response induced by heat exposures is higher heart and respiratory rates to reduce the core body temperature. This response subsequently induces a greater uptake of air pollution into the body. Exposure to temperatures outside of the thermal comfort range may result in loss of the body's ability to regulate and maintain normal function (Witzmann 2009). This in itself can lead to adverse health outcomes, for example, those due to heat stress. In addition, this physiological stress may result in higher susceptibility to and lower defence against toxic substances, including air pollutants. Carder et al. (2008) hypothesised that the stronger risk on mortality in association with black smoke and extremely low temperatures may be related to higher susceptibility to PM_{10} due to the reduction of mucocilliary clearance in the upper respiratory tract induced by cold stress.

Another potential explanation might be related to shared patho-physiological pathways between the two exposures, causing similar adverse health outcomes such as cardiovascular and respiratory illnesses. Ren et al. (2011) studied heart rate variability in response to changes in daily temperature and ambient air pollution in an ageing population. The authors concluded that greater decreases of heart rate variability measures on warm days with high O_3 concentrations were likely to be indicative of temperature and O_3 acting together on the autonomic nervous system. Lin and Liao (2009) provided a similar explanation for their findings of synergistic effects of exposure to high CO concentrations and cold temperatures on cardiovascular mortality. They speculated that the synergy might be a result of increased heart rate and cardiac output and high blood pressure, which were triggered by sharp increases in sympathetic activity and peripheral vasoconstriction to preserve the heat loss.

Laboratory, animal and human studies have rarely been conducted to provide confirmatory evidence suggested by these epidemiological studies or to pin down the biological mechanisms induced by concurrent exposures to the two environmental factors. All the explanations provided by the studies reviewed here on the plausible mechanisms of the joined effects caused by concurrent exposure to air pollution and temperature are hypothesised based on bringing together evidence found from toxicological studies examining solely the health effect of air pollution or temperature.

2.4 The roles of air quality modelling in air pollution and health studies

Epidemiological studies have increasingly utilised pollution data simulated from air quality models for studying the effects of air pollution on health. The use of air quality modelling data can fill gaps that conventional air pollution monitoring measures cannot provide. In this section, two major functions of air quality modelling in air pollution and health studies are explored and discussed. First, data simulated from air quality modelling can help reduce exposure measurement error in estimating dose-response functions. This type of error is common when pollution data from a stationary monitoring network is used. Second, an air quality model can play a role in analysing air quality and related health effects under different scenarios of air pollution emissions.

As the term 'air quality modelling' is broad and may be differently defined and categorised, the review on the roles of air quality modelling in this section is restricted to dispersion and photochemical models. These two types of air quality modelling are widely accepted and used because of their reliability, accuracy and user-friendly for simulations of atmospheric concentrations of primary and secondary air pollutants at urban and larger scales (Daly and Zannetti. 2007).

2.4.1 Reducing exposure measurement error in estimating dose-response functions

Exposure measurement error has been identified as a key issue in air pollution epidemiology (Sheppard et al. 2012; Sarnat et al. 2007). In most time-series studies focusing on impacts of outdoor air pollution on health, personal exposures are assumed to be equivalent to air pollution measurements obtained from central monitoring stations. This is probably due to the fact that this source of air pollution data is easily accessible, highly cost-effective and highly available for a long retrospective period. In this case, measurement errors can be classified into three components: i) the difference between

averaged exposure for individuals estimated using population data and the true individual exposure, ii) the difference between personal and estimated exposures based on measurements and iii) the difference between true ambient concentrations and readings obtained from measurement devices (Zeger et al. 2000). A number of studies have attempted to measure to what extent measurement error would impact on health risk estimates in time-series studies (e.g. Sarnat et al. 2010; Goldman et al. 2010; Goldman et al. 2011; Sheppard et al. 2005). Overall, findings from these studies suggest that statistical significance and the magnitude of risk estimates of air pollution and health could certainly deviate from the true estimates as a result of measurement error, in particular studies of primary air pollutants and PM. The difference between personal and estimated exposure (i.e., component ii, above) has been identified as the largest contributor to bias and is the area in which the use of simulated air pollution concentrations from air quality modelling can play a significant role.

To minimise exposure misclassification, a number of alternative methods to estimate personal exposures to air pollution have been developed. Jerrett et al. (2005) reviewed six different models used to estimate intra-urban exposure for health studies. Among the models reviewed, line dispersion and integrated meteorological emission models have been commonly applied in health effects assessment for community and large scales respectively. For dispersion models, Jerrett et al. (2005) concluded that the greatest advantage in applying this modelling approach was the ability to handle temporal and spatial variability of air pollution at different spatial scales, in both urban and regional settings. Some disadvantages of using the dispersion models were also identified, such as the requirement of intensive data inputs, assumptions on dispersion patterns that deviated from reality, validation of the modelled output with observations and errors from temporal mismatches in the data. For the integrated meteorological emissions model, despite several disadvantages and limited application in health studies, it has some potential benefits over the other models in future work, mainly through: i) simulating multiple possible exposure scenarios and ii) providing more accurate exposure estimates for secondary pollutants including O_3 and secondary PM.

An example of how an ambient air quality modelling system can improve estimations of relative health risks of air pollution by accounting for spatial variability in air pollution exposure, which may lead to reduction of measurement error, was reported in the study by Valari et al. (2011). That study estimated mortality risks associated with exposure to NO_2,

$PM_{2.5}$ and O_3 in the Greater Paris Region, France. A percentage increase in mortality risks for $PM_{2.5}$ and O_3 derived from a multi-pollutant Poisson regression model using modelled concentrations of air pollution exposure combined with county-level data on population density and time-activity was estimated to be greater than that solely based on aggregated monitoring network concentrations. Further, a negative association between $PM_{2.5}$ and mortality estimated using measured air pollution data altered to be positive based on the model using simulated air pollution data, reflecting the true effect of air pollution which is harmful to health. The authors noted that the greater mortality risk estimate for $PM_{2.5}$ yielded based on using the modelled data was due to the better air pollution data at fine resolution provided by the air quality model compared to that using the observed data which were obtained from only one fixed monitoring site over the study domain.

In addition to the advantage of the reduction of measurement error, air quality modelling can be a useful tool to provide fine-scale air pollution exposure that can be used further to detect differences of dose-response functions for air pollution or to estimate various health burdens across areas of a large region such as at the city or country levels. Erbas et al. (2005) investigated variability in estimates of risk for asthma among children living in different urban regions in Melbourne, Australia. To achieve the research aim, an air quality modelling system, TAPM, was used to estimate weather and air pollution exposures for each of four regions in the study area. The study reported that the risk estimates for O_3 and PM_{10} were inconsistent across the urban regions, indicating non-uniform relative risks in an urban setting. Fann et al. (2012) quantified health burdens associated with exposure to ambient $PM_{2.5}$ and O_3 across all the states in the US in 2005. Using concentrations of the pollutants simulated by an air quality modelling system, Community Multiscale Air Quality (CMAQ), the study was able to estimate exposures for the whole country, including locations where ambient air pollution measurements were not available. To further improve the accuracy of the air quality data, the simulated ambient pollution data were combined with measurements from monitoring stations.

2.4.2 Predicting air pollution concentrations under different scenarios

Air quality modelling enables personnel working in air quality management to estimate ambient air pollution levels under different scenarios and assess consequences for health. This role is specifically beneficial for selecting control measures or policies that are both economical and able to protect population health.

Scales of application of air quality modelling for this purpose can range from community to city, regional and national levels. Capon et al. (2008) demonstated this type of application at the community level. The authors simulated air pollution emitted from an exhaust stack of a tunnelled roadway in Sydney using an atmospheric transport and chemistry model. Then, to identify the health impacts, the simulated concentrations were linked with responses to telephone interviews on symptoms likely to be caused by exposure to air pollution. At the city level, Vlachokostas et al. (2009) used air quality modelling in combination with other tools for constructing four air pollution scenarios in a Greek city. Costs of control and health benefits were compared for each scenario to support future decision making. At the national level, Boldo et al. (2011) estimated deaths avoided by the successful implementation of planned $PM_{2.5}$ emissions control measures that would lead to a reduction of 0.7 $\mu g/m^3$ of $PM_{2.5}$ from the 2004 baseline level in Spain. $PM_{2.5}$ concentrations were simulated and linked with other information, including dose-response functions of $PM_{2.5}$ on mortality outcomes, populations at risk and mortality rates for the baseline scenario.

Another vital role for air quality modelling to play in public health is to forecast future ambient air pollution concentrations. For daily forecasting, air quality modelling has been used, similar to weather forecasting, to provide air quality information to the public so that people can plan daily activities to respond to the situation and to protect their health appropriately (Carnevale et al. 2011; Cope et al. 2008a; Zhang et al. 2012). For long-term predictions, in particular effects of climate change on air quality, air quality modelling has been used in combination with climate modelling to predict future air quality. The use of air quality modelling for this purpose and its linkages to predict health impacts will be reviewed and discussed in Chapter 7.

2.5 Chapter summary

The review undertaken in this chapter has provided broad understanding in three areas relevant to Objectives 1 and 2 of this thesis. The first area considered in this chapter related to epidemiological studies of air pollution and health in Australia. Generally, these studies have continuously provided evidence on how and the extent to which air pollution is harmful to Australians. Different study designs and methods have been applied to investigate acute adverse effects of air pollution on health including two national multi-site studies—SPIRT and EPHC. Despite inconsistent findings reported by these studies, a

general conclusion is that air pollution in Australia is still adversely affecting the health of Australians, particularly those suffering from cardiovascular and respiratory diseases.

The next area reviewed in this chapter contributed to shaping the way in which the investigation of interaction effects between temperature and air pollution on health in Melbourne, reported later in Chapter 6, had been set up. Increasingly, more evidence on a temperature effect that modifies the health impacts of air pollution has been reported by epidemiological studies in different locations of the world. In Australia, only a few studies have been conducted to examine how the relationship between air pollution and health would alter across the temperature range. Results from most of these studies suggest that stronger health risks associated with air pollution can be found at both lower and higher ends of the temperature range. Three plausible mechanisms of a synergistic health effect of simultaneous exposure to temperature and air pollution have been commonly used to explain the results reported by these epidemiological studies. Even though the temperature modifying effect seems to be plausible, more studies to understand the mechanisms are required in order to confirm the results from these epidemiological studies.

Last, this chapter discussed the roles that air quality modelling can play in studying the health effects of air pollution. There are two major roles fitting the context of this thesis. The first role is to reduce measurement error in estimating dose-response functions for studying the epidemiology of the health risks of air pollution. The degree to which using air pollution data simulated from an air quality model can improve estimates of the health risks related to air pollution exposure in this thesis will be explored in Chapter 5. The other application of air quality modelling in health research is to predict air quality under different scenarios. Using an air quality model in this thesis to estimate health impacts associated with air pollution as a result of climate change will be reported in Chapter 8.

Chapter 3 Data description

3.1 Introduction

As reviewed in the previous chapter, conducting a time-series study in air pollution epidemiology requires three basic types of data, namely: health, air pollution and weather. This thesis uses these data to estimate relative risks of acute health outcomes related to air pollution, while taking into account interactions between temperature and air pollution. This chapter displays key characteristics of these datasets through descriptive statistics and exploratory analyses.

The first two sections in this chapter provide details of data sources and how the data were processed to prepare them for analysis. The following section provides descriptive statistics and exploratory analyses of health outcomes of interest, air pollution and weather in the study area during the period of interest. Trends and patterns of each of these datasets over time are first explored and described individually. Next, correlations within and between these datasets are also examined and presented.

The air pollution and weather data described in this chapter are based on readings obtained from monitoring stations in the study area. Details of combined simulated and measured datasets are in the next chapter.

3.2 Data sources

Data on daily health outcomes of interest, air quality and weather data were collected for the period 1999 to 2008. The boundary of this study was limited at SD 205—Melbourne Region in Victoria—according to the 2008 ASGC. This area includes 79 SLAs as mentioned in Chapter 1. Mortality data were obtained from a restricted database held at the National Centre for Epidemiology and Population Health (NCEPH). The original custodian of this dataset was the Victorian Registrar of Births, Deaths and Marriages. Morbidity data, here ED visits, were supplied by Victorian Department of Health. Ambient air quality data were sourced from the Environmental Protection Authority (EPA) Victoria. Although there were 22 monitoring stations, permanent and short-term, located in Victoria during the study period, this thesis used only the readings from the 14 stations that are located in SD 205 that is the study area (see Figure 3.1). The eight stations excluded are all located at some distance from the study area and its borders (see Appendix A.1) so that the observed data do not contribute relevant data to the population exposure to air pollution within the study area.

Weather data were derived from the Australian Bureau of Meteorology (BoM). Although there were 14 weather stations in operation during the period of interest, the data for this study were restricted to nine automatic weather stations (AWSs) with hourly recording of data. The rationale for limiting to the AWSs was to provide consistency of input weather data for two methods applied in this study to estimate personal exposures at the SLA level, including: i) proximity estimates based on measurements from the nearest station and ii) blending of measurements with gridded fields simulated by a meteorological modelling system. The details of the second method are given in Chapter 4. The locations of the nine weather stations are shown in Figure 3.1.

Since the ambient air quality monitoring stations also measured dry-bulb air temperatures, this study also used temperature measurements from EPA monitoring stations. Therefore, temperature readings were from 25 stations (nine AWSs and 14 ambient air quality stations), while dew point temperature readings were from nine AWSs.

Figure 3.1 Fourteen ambient air monitoring stations (green dots) and nine AWSs (yellow dots) located within SD 205 during 1999 to 2008

3.3 Data preparation

The three datasets were initially prepared so that analyses at the spatial unit of SLA level could be made. There were two minor changes to SLA codes during 1999 to 2008. The first change occurred in 2000 and the other in 2004. Therefore, in the data preparation

stage, six 1999–2004 SLAs (see Appendix A.2) were standardised to be compatible with the 2005–2008 SLAs.

In line with the SLA change, the daily mortality/morbidity counts of the three SLAs (namely, 7074, 3671 and 7455) for the period 1999 to 2004 were split and assigned to the 2005–2008 SLAs accordingly. Two of these (7074 and 3671) were each split into two SLAs. The other, 7455, was split into three SLAs. The 2006 population representing the population during 2005 to 2008 was used to calculate percentages of the daily mortality/morbidity counts of the split seven 2005–2008 SLAs, applying retrospectively to those estimates in the original three 1999–2004 SLAs (see Table 3.1).

For each SLA, the weather and air pollution data were taken from the monitoring station nearest to the centroid of it. Since the number of monitoring stations in operation was variable each year, only the stations that had readings for over 75% of days for each variable were included in the study. The number of monitors excluded varied across the study years and for the different pollutants, with 6–8 excluded for $PM_{2.5}$ and 3–7 excluded for O_3.

Table 3.1 Percentages used to estimate the 1999 to 2004 daily mortality/morbidity counts for the SLAs 7074, 3671 and 7455 to match with the 2005 to 2008 SLAs

1999–2004 SLA	Corresponding 2005 to 2008 SLA	2006 population based on 2005 to 2008 SLA	2006 population percentage applied to calculate daily counts for 1999 to 2004 SLA
7074	7075	44 374	42.1
	7076	61 154	58.0
3671	3672	64 256	58.0
	3673	46 463	42.0
7455	7452	30 452	26.2
	7453	70 029	60.2
	7456	15 787	13.6

3.4 Data description

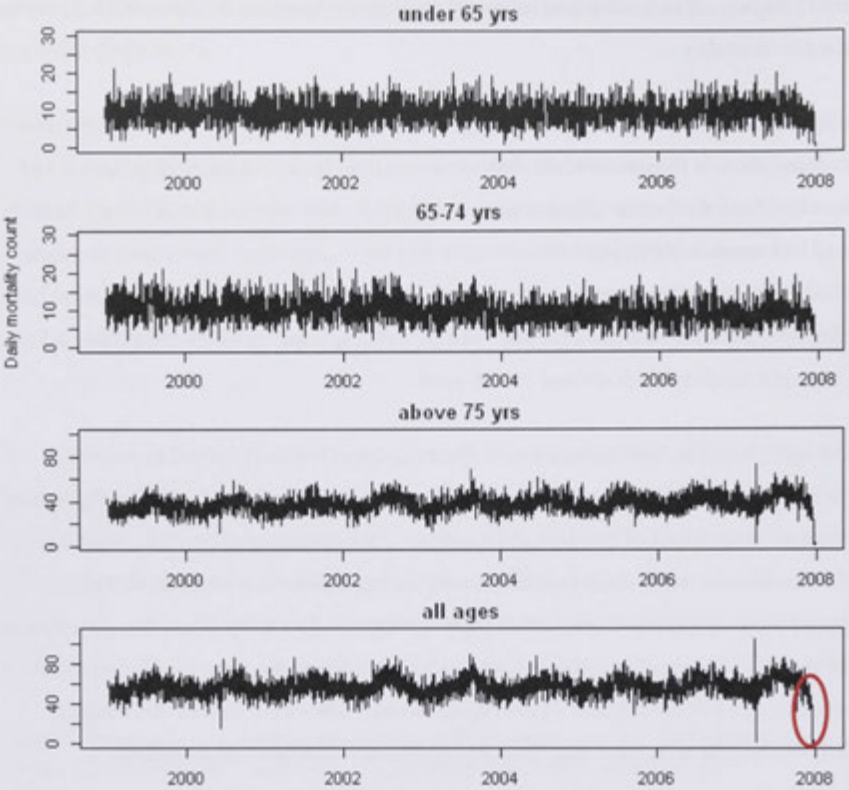
3.4.1 Health data

The description of health data presented in this chapter follows the ICD-10 diagnostic codes. Mortality was classified into three classes, including all-cause (ICD-10 A00-R99), cardiovascular (ICD-10 I00-I99) and respiratory (ICD-10 J00-J99). ED visits were

classified into two classes, cardiovascular and respiratory, by using the same ICD-10 codes as applied to mortality.

Mortality data were included from 1 January 1999 to 31 December 2007. For the purpose of data description in this section, the data were stratified by ages into three groups—<65, 65–74 and >75—for all of the disease classes. ED visits data were included from 1 January 1999 to 31 December 2008. For cardiovascular ED visits, age-stratification was the same as applied to the mortality data. In order to also explore respiratory illness in children, ED visits data for this disease class were stratified into four groups, in which the group <65 years was split further into 0–14 and 15–64 years.

It can be seen from the time-series plots in Figure 3.2 that the daily mortality counts towards the end of 2007 fell sharply, such as those in the red circle. When checking, it was found that more than half of the days in December 2007 contained daily events that were much lower than the mean daily mortality count during the entire study period. The anomaly of these daily counts may result from incomplete data entry. Therefore, the data in December 2007 (737 death counts) are not further included in the analyses performed in Chapters 5 and 6. For the purpose of providing an overview of the data in this chapter, however, all the mortality data over the study period are included and described.



Note: The red circle indicates a sharp fall of daily death counts towards the end of 2007

Figure 3.2 Time-series plots of all-cause mortality by age, January 1999 to December 2007.

3.4.1.1 Descriptive statistics for the health data

Descriptive statistics for the health data are shown in Table 3.2. Cardiovascular and respiratory disease of Melbourne residents followed the typical pattern. Large mortality and morbidity counts for circulatory disease were predominantly found in the elderly aged over 65, while morbidity counts for respiratory disease were dominant at ages under 65. For patients aged <65 years who were ill from respiratory disease, the group aged below 15 years was the largest for the ED visits category.

An outlier was defined as a data value exceeding $\pm$ 5 times the standard deviation. Outliers for the health data were as follows. For the mortality outcomes, there were outliers in the data for three outcomes: all-cause mortality for all ages (14 outliers), respiratory mortality

at age under 65 (four outliers), and respiratory mortality aged over 75 (one outlier). For example, within the data on all-cause mortality for all ages, on 25 March 2007, the daily mortality count was 104 (+5 standard deviation = 103) .In the remaining 13 outliers, the daily mortality counts were below 9 (-5 standard deviation = 9), and twelve of these occurred in December 2007. This example supports the decision to remove the mortality data from December 2007 from the analyses in Chapters 5 and 6, as previously noted. For the ED visits outcomes, two outliers larger than 130 daily counts (+5 standard deviation) were found for the respiratory ED visits in the age group 15-65 years.

3.4.1.2 Trends and patterns of the health outcomes of interest

Long-term trends and seasonal patterns over time for the health outcomes of interest were examined based on time-series plots. The discussion in this section focuses on two disease classes, circulatory and respiratory disease, and also all-cause mortality.

There were no visible temporal trends in all-cause mortality, regardless of age group, over the years of the study period (see Figure 3.2). This was similar for circulatory and respiratory disease mortality (see Appendices A.3a and A.3b). Seasonal patterns were apparent for all-cause, as well as for both circulatory and respiratory disease categories. The strongest pattern was noticeable in the >75 years group, possibly because it was the largest age group. Figure 3.3 demonstrates a seasonal pattern of all-cause mortality for the period 1999 to 2001 for all ages. Typically, the mortality peaked in winters (June to August) and dropped in summers (December to February).

Figures 3.4 and 3.5 show trends of increased daily counts of cardiovascular and respiratory ED visits respectively over the study period. To some extent, these trends might result from an increase in the size of population in the Melbourne Region. Typical seasonal patterns of cardiovascular and respiratory ED visits were also found as previously described for mortality data in which the daily morbidity counts were highest in winters and lowest in summers. The seasonal patterns were more obvious for respiratory ED visits, compared to circulatory ED visits.

Although seasonal patterns of cardiovascular and respiratory disease are observed in many parts of the world, underlying mechanisms have not been well understood, particularly for cardiovascular disease. The seasonal patterns described here for cardiovascular mortality and ED visits are consistent with a study in Australia conducted by Barnett et al. (2008).

That study demonstrated that the more temperate a city, the stronger is the peak of cardiovascular mortality in winter. Two predisposing causes—both driven by cold temperature in winter—including increased blood pressure and lack of vitamin D, were used to explain in that study why cardiovascular disease was highest in winter. Another study in Norway hypothesised that the peak of cardiovascular disease in winter may be associated with temperature and relevant behaviours that could consequently increase risk factors of cardiovascular disease such as body weight, cholesterol levels and blood pressure (Hopstock et al. 2013). That study found statistical significance of seasonal patterns of most of the risk factors examined with the peak in winter, indicating that these factors might be used to explain the seasonal pattern of cardiovascular disease. However, the magnitude of the seasonal change of these risk factors was not very strong, which may be due to adaptability of people to the cold climate in that region.

A number of studies have investigated factors driving the high incidence of respiratory disease in humans in winter. Such high incidence in winter is apparent in both infectious and non-infectious respiratory diseases. For respiratory infections such as influenza, evidence from an experimental study, from a perspective of viral agents, suggested that low temperature and low humidity in winter increased the transmissibility and survival of an influenza virus (Lowen et al. 2007). From a perspective of the host, that study explained that breathing dry and cold air in winter could reduce defence mechanisms in the upper respiratory tract such as inhibiting the mucociliary clearance function. For non-infectious respiratory disease such as COPD, strong seasonal patterns of COPD exacerbations have also been reported in winter, predominantly in patients living in temperate regions (Jenkins et al. 2012; de la Iglesia Martinez et al. 2000). The increased of rate in COPD exacerbations in winter may be explained by increased susceptibility of COPD patients to respiratory infections and resulting reduced lung function (Donaldson et al. 1999; Jenkins et al. 2012).

Table 3.2 Descriptive statistics for daily counts of health outcomes by age category, 1999–2008

Health outcome	Total counts over the study period	Mean of daily counts	Standard deviation of daily counts	Minimum of daily counts	Maximum of daily counts
Mortality*					
All-cause					
All ages	184 730	56.32	9.43	0	104
<65 yrs	30 724	9.37	3.09	0	21
65–74 yrs	31 347	9.57	3.26	0	21
≥75 yrs	122 659	37.37	7.71	0	72
Cardiovascular					
All ages	68 414	20.88	5.31	1	42
<65 yrs	6 351	1.94	1.40	0	8
65–74 yrs	8 928	2.73	1.77	0	10
≥75 yrs	53 135	16.22	4.49	1	36
Respiratory					
All ages	16 066	4.90	2.42	0	17
<65 yrs	1 199	0.37	0.61	0	4
65–74 yrs	2 458	0.75	0.87	0	5
≥75 yrs	12 409	3.78	2.11	0	15
ED visits					
Cardiovascular					
All ages	358 074	98.02	15.86	51	155
<65 yrs	154 771	42.37	8.60	18	73
65–74 yrs	73 942	20.24	5.03	5	40
≥75 yrs	129 361	35.41	8.21	13	72
Respiratory					
All ages	641 601	175.64	48.15	66	405
0–14 yrs	303 969	83.21	31.24	16	226
15–64 yrs	199 612	54.64	15.11	19	239
65–74 yrs	48 933	13.40	4.59	1	32
≥75 yrs	89 087	24.39	8.43	5	56

Note: * The mortality data are for the period 1999 to 2007

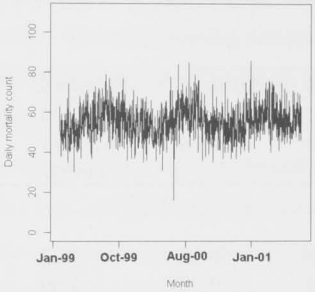


Figure 3.3 Seasonal pattern of all-cause mortality for all ages, January 1999 to December 2001

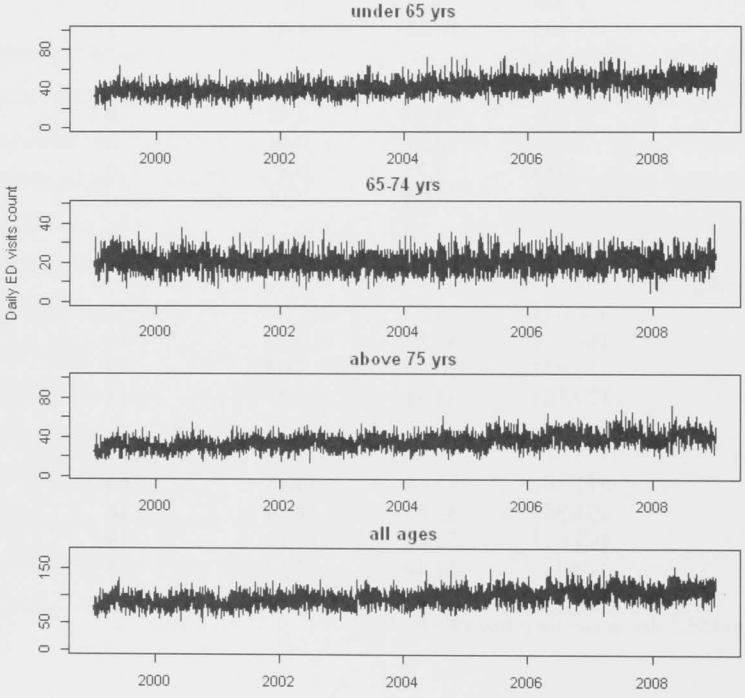


Figure 3.4 Time-series plots of cardiovascular ED visits by age, January 1999 to December 2008

3.4.2 Air quality data

Air quality data presented in this section include two air pollutants of interest—O_3 and $PM_{2.5}$. Bsp data are also presented here, as they were used throughout this study as surrogates for $PM_{2.5}$ on the days that $PM_{2.5}$ measurements were not available. Using Bsp as a surrogate for $PM_{2.5}$ is a common practice in other air pollution and health studies in Australia where readings on $PM_{2.5}$ were not available before 2002 (Simpson et al. 1997; Petroeschevsky et al. 2001; Jalaludin et al. 2006). Some points should be noted in relation to the $PM_{2.5}$ measurements used in this thesis. In Victoria, $PM_{2.5}$ is measured by two methods, Tapered Element Oscillating Microbalance (TEOM) and Partisol.

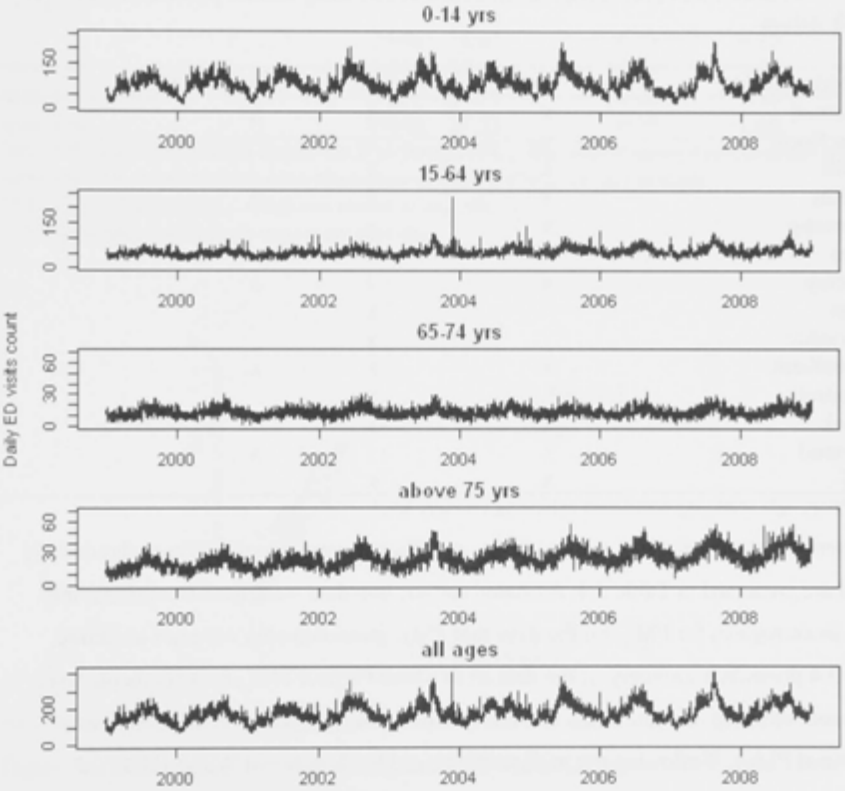


Figure 3.5 Time-series plots of respiratory ED visits by age group, January 1999 to December 2008

This thesis used only measurements from Partisol, as it is the reference method specified in the Ambient Air Quality National Environmental Protection Measure (AAQ NEPM)

(Environment Protection and Heritage Council 2003). In comparison with TEOM, this method provides more accurate measurements due to its ability to preserve volatile components during the processing of samples (Charron et al. 2004). Monitoring Partisol $PM_{2.5}$ in Victoria was commenced in July 2002 as part of the process to introduce $PM_{2.5}$ standards later enforced in the 2003 variation to the AAQ NEPM. For Partisol $PM_{2.5}$, samples are taken and measured once every three days. Table 3.3 shows the air pollutants of interest measured at each of the 14 monitoring stations located in SD 205. Among the 14 stations, two were campaign stations operating for only a short period (Eltham for the period 2005 to 2006 and Mooroodue for the period 2004 to 2006).

Table 3.3 Fourteen monitoring stations included in this study and air pollutants measured at each station

Monitoring station	Bsp*	O_3	Partisol $PM_{2.5}$
Alphington	x	x	x
Altona North	x	x	
Box hill	x	x	
Brighton	x	x	x
Dandenong	x	x	
Eltham		x	x
Footscray	x	x	x
Melton		x	
Mooroodue		x	
Mooroolbark	x	x	x
Mt.Cottrell		x	
Pt.Cook	x	x	
Richmond	x		x
RMIT	x	x	

Note: *Bsp; light scattering coefficient indicating visibility levels

Descriptive statistics for Bsp, O_3 and $PM_{2.5}$ at different averaging times throughout study period are presented in Table 3.4. As stated earlier, Bsp data were used throughout this thesis as surrogates for $PM_{2.5}$ on the days that $PM_{2.5}$ measurements were not available. Table 3.4 presents a summary of the dataset in which Partisol $PM_{2.5}$ measurements were combined with Bsp measurements and converted to $PM_{2.5}$; this is referred to here as 'combined $PM_{2.5}$'. Following the method developed by Wendt and Walsh (2007), a regression function of Bsp and Partisol PM $_{2.5}$ was developed by exploiting samples at the Alphington site during the period 2002 to 2008 to estimate $PM_{2.5}$ based on Bsp measurements as shown in Figure 3.6.

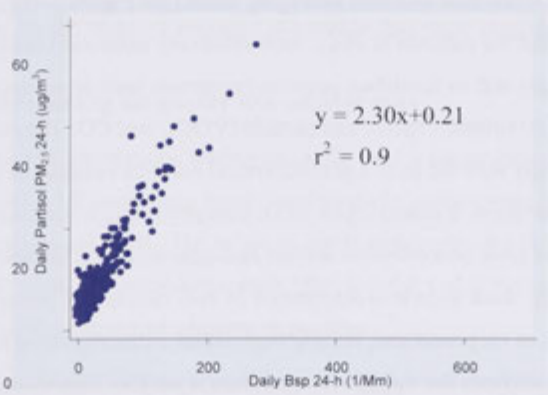
Table 3.4. Descriptive statistics for daily concentrations of Bsp, O_3, Partisol $PM_{2.5}$ and combined $PM_{2.5}$* (network average) in the Melbourne Region, January 1999 to December 2008**

	Number of measurements	Mean	Standard deviation	Minimum	Maximum
Bsp 24-h (1/Mm)	3 648	24.55	31.92	4.21	698.69
Partisol $PM_{2.5}$ 24-h ($\mu g/m^3$)***	961	7.74	5.88	0.40	52.15
Combined $PM_{2.5}$ 24-h ($\mu g/m^3$)	3 648	7.42	6.64	3.00	147.63
O_3 (ppb)					
24-h	3 649	14.61	6.18	0.14	46.40
max 1-h	3 649	27.52	10.86	2.87	109.00
max 4-h	3 649	25.77	10.04	1.50	100.33
max 8-h	3 648	23.21	9.25	1.00	85.63

Note: * Primary source of this dataset was from Partisol $PM_{2.5}$; Bsp concentrations were calculated to obtain additional $PM_{2.5}$ concentrations when the samples of Partisol $PM_{2.5}$ were not available.

** Except for Partisol $PM_{2.5}$, which was started in July 2002

*** Measurements were made once every three days



Note: Data recoded between 29 July 2002 to 31 December 2008

Figure 3.6 Relationship of Partisol $PM_{2.5}$ and Nephelometer Bsp at Alphington

Although the relationship between Bsp and $PM_{2.5}$ may vary temporally, the coefficient of determination (r^2) between these two variables of 0.9 (as shown in Figure 3.6) which is considered very high indicates a slight variation of the relationship over time. Notably, this thesis did not rely only on measured $PM_{2.5}$ derived from Partisol $PM_{2.5}$ and Bsp. Rather this thesis used a blending of the measured and modelled data. The blending method

employed, therefore, has the potential to minimise the impact of the variation of the relationship between Bsp and $PM_{2.5}$ over time.

Using the same definition of an outlier as applied to the data of health outcomes previously described, the number of outliers for air pollution variables (network averages) as displayed in Table 3.4 for Bsp, Partisol $PM_{2.5}$ 24-h, combined $PM_{2.5}$ 24-h, and the four O_3 variables were 21, 6, 21, 1, 6, 5 and 5 respectively. The dates that contain outliers for Bsp (>184.15 1/Mm) and combined $PM_{2.5}$ 24-h (>40.64 $\mu g/m^3$) are identical. Furthermore, all of these days with the exception of one day on 12 Apr 2000 are those identified to have exceedences of PM_{10} or $PM_{2.5}$ caused by bushfire events (see Appendix A.4a). Three out of the six outliers for Partisol $PM_{2.5}$ were found on the days when PM_{10} or $PM_{2.5}$ exceeded the standards due to bushfires. Apart from one outlier for O_3 24-h and one outlier for O_3 max 1-h, all of the outliers for O_3 variables were found on bushfire days as shown in Appendix A.4a. These outliers that were associated with bushfire events were removed and not further used in the analyses.

Several air pollutants typically exhibit seasonal patterns (Peng and Dominici 2008). In this thesis, monitored O_3 levels at different averaging times (see Figure 3.7) clearly showed seasonal patterns, but the patterns of $PM_{2.5}$ were relatively indistinct (see Figure 3.8). O_3 is a secondary pollutant and its formation requires precursors such as oxides of nitrogen (NO_x, NO_2 and NO), volatile organic compounds (VOCs) and CO. The reaction of O_3 formation is generally referred to as a photochemical reaction because of the requirement of sunlight. In urban areas, a diurnal cycle of O_3 concentrations is common, which is characterised by the peak concentration around mid-afternoon and the lowest concentration in the early morning. Such a cycle is determined by two factors: the presence of sunlight and high emissions of O_3 precursors, mainly from motor vehicles during rush hours (Vallero 2008). Apart from the sunlight, temperature is another important catalyst of O_3 because it increases emissions of VOCs, particularly from biogenic sources, which is one of the major O_3 precursors (Jacob and Winner 2009). Hence, O_3 concentrations, particularly in temperate climate conditions, are noticeably higher in summers than in winters.

For $PM_{2.5}$, the lack of a distinct seasonal pattern may be due to the different emissions sources of the pollutant, which are not necessarily seasonal. For example, the most extreme values of $PM_{2.5}$ were associated with bushfires, which often occurred in summer (see

Appendix A.4a). However, high levels of $PM_{2.5}$ were sometimes apparent in cooler months, in association with the use of wood heaters. Alternatively, naturally occurring dust episodes (see Appendix A.4b) and anthropogenic planned burns do not have strong seasonality, with the latter occurring throughout the year when conditions are suitable (EPA Victoria 1999-2008).

3.4.3 Weather data

Weather variables are commonly included in epidemiological models of air pollution and health as they are able to confound and modify the health risk estimates (Samet et al. 1998). In this thesis, two weather variables included in epidemiological models are dry-bulb temperature and dew point temperature. Descriptive statistics of daily weather data presented in Table 3.5 were generated from averaging readings across available monitoring stations located in the study area on a particular day (network average) before averaging them again over the study period. Time-series plots of the weather variables of interest show clearly seasonal patterns (see Figure 3.9). Temperatures were highest in summers (December to February) and lowest in winters (June to August). With the application of the same definition of an outlier to the weather variables (±5 standard deviation) from the mean), no outliers for the network averages of weather data were identified.

3.4.4 Correlations among air quality and weather data

In this section, Pearson correlations among air quality and weather data are explored (see Appendix A.5). Table 3.6 summaries these correlations by categorising them into three levels: strong, medium and weak. The levels of correlation were considered strong, medium and weak when the correlations were >0.8, 0.4–0.8 and 0–0.4 respectively. The directions of correlations could be negative or positive.

As discussed earlier in Section 3.4.2, Bsp and Partisol $PM_{2.5}$ are highly positively correlated. Due to the fact that *combined* $PM_{2.5}$ data were generated from Bsp and Partisol $PM_{2.5}$, these pairs have undoubtedly strong positive correlations. O_3 has generally weak correlations, either positive or negative, with $PM_{2.5}$. This can be explained by considering emissions sources of $PM_{2.5}$ and O_3 precursors in the PPR. According to Victoria Air Emissions Inventory 2006, the largest source of O_3 primary precursors, NO_x, was from motor vehicles (~60%), while major sources of PM were from fires (~25%) and solid fuel combustion (~15%). $PM_{2.5}$ emitted from motor vehicles contributed only 10% (EPA

Victoria and CSIRO 2007). However, the correlations of O_3 and PM parameters vary by season. In summer, the correlations become higher at the medium positive level (see Appendix A.5b). This may be to a certain extent related to the occurrence of bushfires in summer, which not only releases PM but also O_3 precursors, including NOx, non-methane VOCs and methane. O_3 exceedences were occasionally found during bushfires (EPA Victoria 1999-2008). In winter, O_3 and $PM_{2.5}$ are negatively correlated in the medium range. As stated in Section 3.4.2, by the nature of photochemical reactions and the increased formation at high temperatures, O_3 concentrations in cooler months are low. In contrast, lower temperatures in winter result in increased use of wood heaters and thus higher PM concentrations.

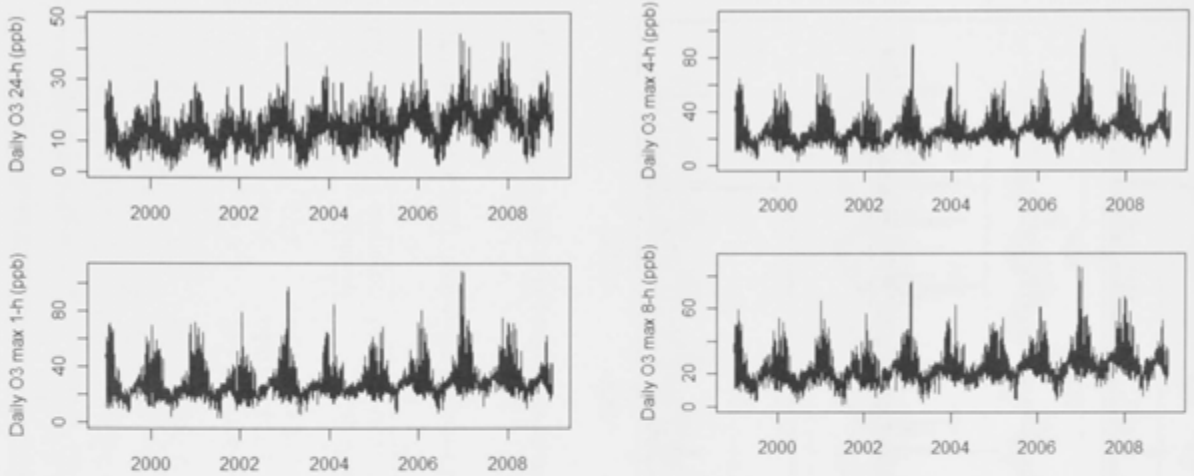


Figure 3.7 Time-series plots of daily O_3 concentration, including 24-h, max 1-h, max 4-h and max 8-h, January 1999 to December 2008

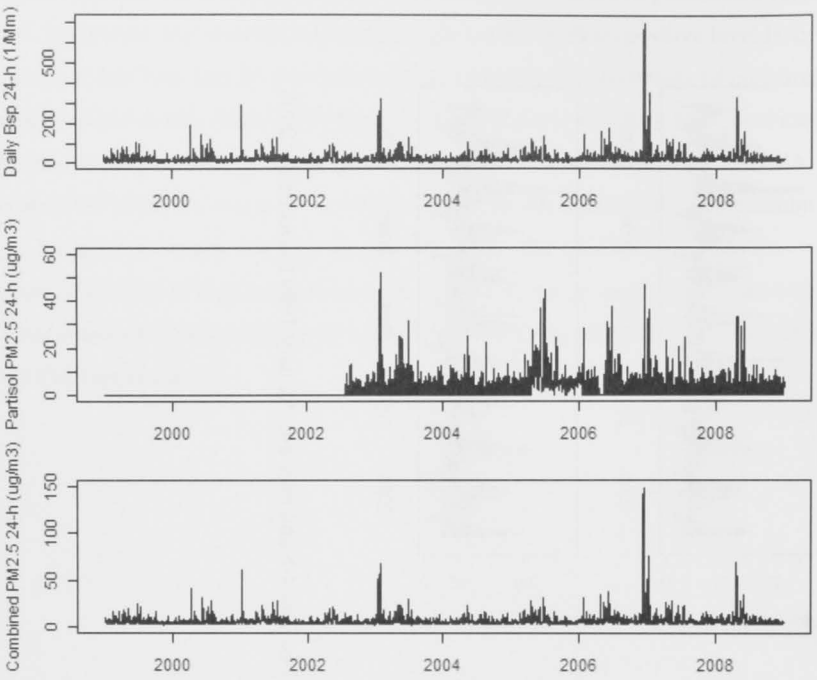


Figure 3.8 Time-series plots of daily Bsp 24-h, Partisol $PM_{2.5}$ 24-h and combined $PM_{2.5}$ 24-h, January 1999 to December 2008

Table 3.5 Descriptive statistics for daily weather data, January 1999 to December 2008

Weather variable	Total measurements	Mean	Standard deviation	Minimum	Maximum
Dry-bulb temperature (°C)					
Average	3 653	14.80	4.63	5.55	33.07
Min	3 653	10.19	3.92	-0.31	25.25
Max	3 653	19.82	6.18	8.27	43.23
Dew point temperature (°C)					
Average	3 645	8.24	3.38	-1.16	20.66

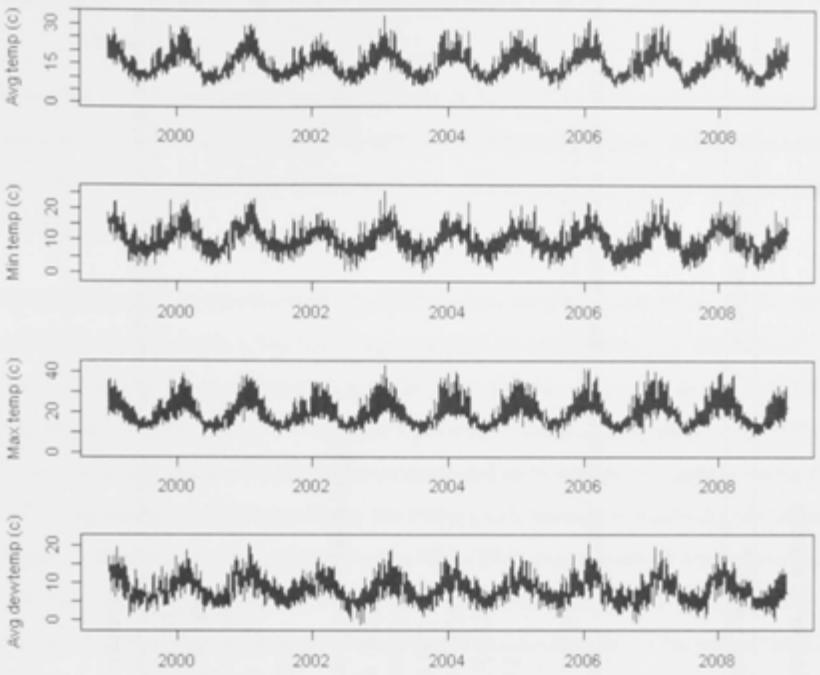


Figure 3.9 Time-series plots of daily average, minimum, maximum of dry-bulb temperature and daily average of dew point temperature, January 1999 to December 2008

Generally, correlations of O_3 and temperature are moderately positive. However, these correlations are stronger, particularly between maximum O_3 variables and dry-bulb temperature in summer, and weaker in winter. These correlations can be clearly explained by the O_3 formation processes in high temperatures. The $PM_{2.5}$ parameters have weak correlations with the weather variables. In winter, their correlations with the minimum dry-bulb temperature variable become stronger, which is likely to be related to smoke emitted from wood heaters on cold days (see Appendix A.5d). Correlations between the dew point temperature variable and other dry-bulb temperature variables are positively moderate (see Table 3.6). However, the correlations of maximum dry-bulb temperature and dew point temperature became weaker when the data were analysed by season; the correlation in the winter was the weakest (0.23), compared to the other seasons (see Appendix A.5).

Table 3.6 Summary of Pearson correlations of air quality and weather variables for all seasons

Pearson correlation	Air quality variable[a]	Air quality and weather variable[a]	Weather variable[a]
Strong positive (r>0.8)	Bsp—$PM_{2.5}$ (p)* (0.82) Bsp—$PM_{2.5}$ (c)** (1.00) $PM_{2.5}$ (p)—$PM_{2.5}$ (c) (0.85)		
Medium positive (0.4<r>0.8)		O_3 (all averaging times[b])—temp max, min and average (0.71, 0.39, and 0.63)	Dew point—temp max, min and average (0.59, 0.79, and 0.70)
Weak positive (0<r<0.4)	Bsp—O_3 (all averaging times[b]) (0.28) $PM_{2.5}$ (p)—O_3 max 1-h (0.08) $PM_{2.5}$ (p)—O_3 max 4-h (0.03) $PM_{2.5}$ (c)—O_3 (all averaging times[b]) (0.27)	Bsp—temp max (0.15) Bsp—temp average (0.07) $PM_{2.5}$ (p)—temp max (0.08) $PM_{2.5}$ (c)—temp max (0.14) $PM_{2.5}$ (c)—temp average (0.07) $PM_{2.5}$ (c)—dew point (0.07) O_3 (all averaging times[b])—dew point (0.29)	
Weak negative (-0.4<r<0)	$PM_{2.5}$ (p)—O_3 max 8-h (-0.04) $PM_{2.5}$ (p)—O_3 24-h (-0.20)	Bsp—temp min (-0.03) $PM_{2.5}$ (p)—temp min (-0.13) $PM_{2.5}$ (p)—temp avg (-0.03) $PM_{2.5}$ (p)—dew point (0.00) $PM_{2.5}$ (c)_temp min (-0.03)	

Note: [a] The number in the parenthesis for each pair of the variables is the exact value of Pearson correlation. [b] The exact Pearson correlations presented in the parenthesis are only for O_3 max 1-h. The exact Pearson correlations for the other averaging times of O_3 are shown in Appendix A.5

*$PM_{2.5}$ (p); Partisol $PM_{2.5}$, **$PM_{2.5}$ (c); combined $PM_{2.5}$

3.4.5 Distributions of the health outcomes and air pollution concentrations by temperature

Since the first component of this thesis focuses on variation of associations between the acute health effects and air pollution across the temperature range, this section primarily provides a description of the health outcomes of interest and air pollution concentrations by temperature.

It was apparent that the observed air pollution data used in Component 1 of this thesis exhibited clear patterns when they were stratified by temperature. On the basis of percentiles of average dry-bulb temperature, as shown in Table 3.7, the level of $PM_{2.5}$ 24-h was relatively high at the 5th temperature percentile and gradually declined at higher temperature percentiles. At the upper extreme end of temperature, approximately from the 75th temperature percentile onwards, the $PM_{2.5}$ levels seemed to gradually elevate again. Figure 3.10 shows a similar pattern when $PM_{2.5}$ 24-h concentrations were stratified into 1°C bins of average dry-bulb temperature. It can be seen from the Lowess (locally weighted scatter plot smoothing) curve fitted in the distribution of the mean $PM_{2.5}$ 24-h concentrations in each bin of temperature range that $PM_{2.5}$ levels tended to be high at low temperatures and gradually decreased along with rising temperatures. The $PM_{2.5}$ 24-h levels were then fairly stable in the temperature range of 15°C to 25°C before increasing again at the temperatures above 25°C. The slope of the $PM_{2.5}$ 24-h concentrations at high temperatures appeared to be greater than that at low temperatures. As explained in the previous section, the high concentrations of $PM_{2.5}$ at the low temperature range in the Melbourne Region are largely related to use of wood heaters and temperature inversion in winters. However, the high concentrations of $PM_{2.5}$ at the high temperature range are partly contributed by the high occurrence of bushfires episodes in summers. The clear pattern of O_3 concentrations across the temperature range was indicative of the higher temperature the increased O_3 concentrations. The slope of O_3 concentrations changed noticeably to be steeper at the temperatures above 15°C.

Table 3.7 Distributions of the health outcomes and air pollutants of interests at different percentiles of 24-h average dry-bulb temperature

Parameter	Percentile of average dry-bulb temperature				
Mean (SD)	5th	25th	50th	75th	95th
24-h avg dry-bulb temp	8.5	11.2	14.3	17.7	24.0
(°C)	(0.1)	(0.0)	(0.1)	(0.1)	(0.3)
$PM_{2.5}$ 24-h	9.1	7.2	6.5	6.6	10.6
($\mu g/m^3$)	(6.2)	(3.4)	(4.6)	(2.3)	(10.7)
O_3 max 1-h	21.5	22.3	22.5	28.0	48.5
(ppb)	(7.1)	(5.5)	(5.7)	(9.1)	(13.2)
O_3 max 4-h	20.0	21.0	21.0	26.2	44.7
(ppb)	(7.5)	(5.7)	(5.5)	(8.1)	(11.9)
O_3 max 8-h	17.4	18.1	18.9	24.3	39.7
(ppb)	(7.4)	(5.8)	(5.4)	(7.5)	(10.4)
All-cause mortality	63.1	58.8	59.5	56.6	55.8
(count)	(8.1)	(9.8)	(11.8)	(7.9)	(6.1)
Cardiovascular mortality	24.6	22.7	21.2	19.8	20.3
(count)	(5.5)	(5.6)	(6.2)	(5.2)	(4.1)
Respiratory mortality	5.8	5.2	5.4	4.5	4.4
(count)	(2.6)	(2.5)	(2.4)	(1.7)	(2.2)
Cardiovascular ED visits	100.0	98.4	97.0	93.9	93.4
(count)	(15.9)	(13.5)	(14.3)	(14.9)	(15.6)
Respiratory ED visits	223.0	194.0	159.8	156.9	142.3
(count)	(45.5)	(39.2)	(31.0)	(36.3)	(27.0)

There were mixed patterns of distributions of the health endpoints of interest across the temperature range. Overall, both daily counts of mortality and morbidity outcomes were high at low temperatures and progressively decreased along with increased temperatures. Of the health outcomes studied, the distribution of only all-cause mortality outcome followed one of the typical patterns—V-shape—of temperature-health relationships as reported in other studies (Yu et al. 2012). According to the V-shape, the number of daily events of all-cause mortality was modest at a certain temperature zone, shown in Figure 3.10 around 20°C. The mortality below or above this temperature zone was higher; the farther away from this zone, the higher mortality. Further, it appeared that the V-pattern of the all-cause mortality data had a larger slope at low temperatures than those at high temperatures.

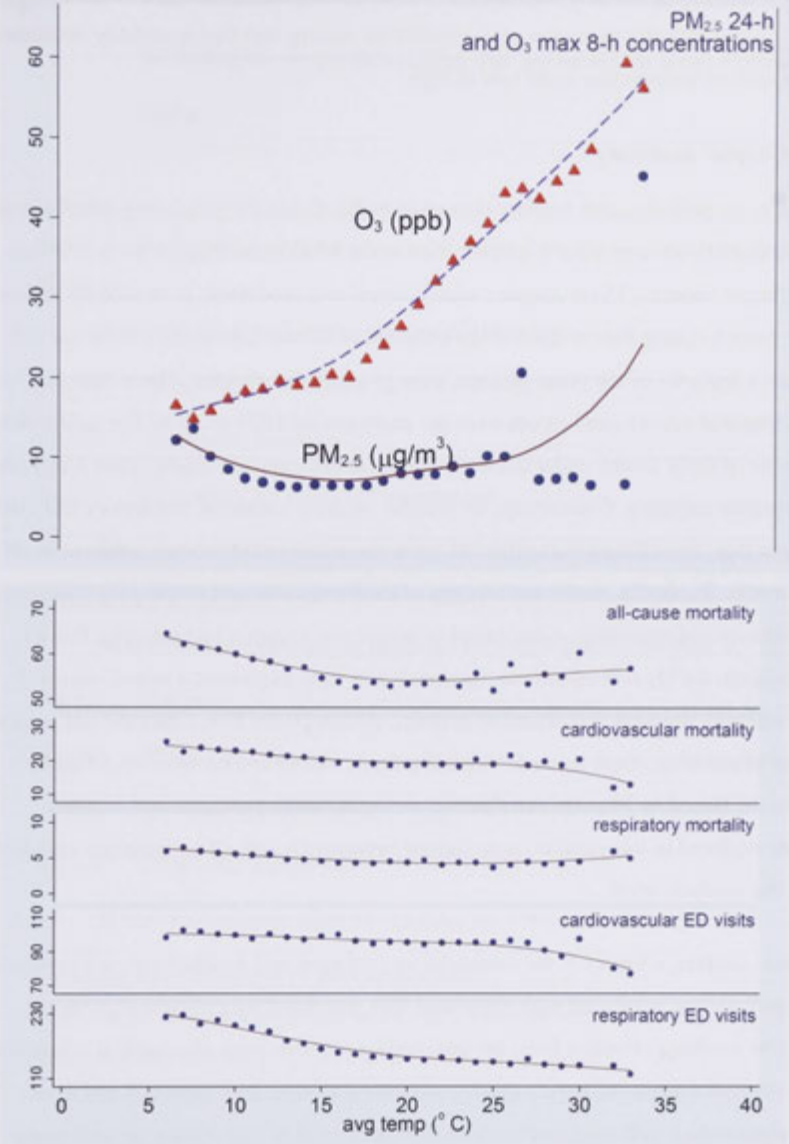


Figure 3.10 Means (scatter dots/triangles) and Lowess lines fitted to the distributions of $PM_{2.5}$ 24-h ($\mu g/m^3$), O_3 max 8-h (ppb) concentrations and the number of daily counts of the health outcomes of interest (all-ages group) in the Melbourne Region across 1°C ranges of average dry-bulb temperature

The pattern of distributed cardiovascular mortality across the temperature range was similar to that of ED visits. For this pattern, the daily counts of cardiovascular mortality and morbidity outcomes gradually decreased with warmer temperatures. It then dropped

abruptly at the average temperatures above 30°C. For the daily counts of respiratory mortality and morbidity outcomes, the distribution pattern was that it steadily decreased with the shift of temperature from low to high.

3.5 Chapter summary

The health, air pollution and weather data used in this thesis for estimating relative health risks associated with exposure to air pollution in the Melbourne Region were obtained from different sources. These datasets were cleaned and processed, as described in this chapter, prior to being further used in the chapters to follow. Descriptive statistics and exploratory analyses of the three datasets were given in this chapter. These data exhibited certain temporal patterns and trends over the study period 1999 to 2008. For health data, the number of daily deaths under the cardiovascular category was higher than that under the respiratory category. Conversely, the number of daily counts of respiratory ED visits was larger than that of cardiovascular ED visits. Seasonal patterns were apparent in all three datasets. Typically, peaks and troughs of cardiovascular and respiratory outcomes, both mortality and morbidity, were found in winter and summer respectively. For air pollution data, the O_3 concentrations measured over time expressed a strong seasonal pattern whereby the peak was found in summer. Although the $PM_{2.5}$ data did not exhibit any clear seasonality, there was a noticeable pattern when the data were stratified by temperature. Based on Pearson correlations, between the air pollution and weather variables explored in this chapter, correlations between O_3 and all temperature variables were at the medium level.

In the next chapter, Chapter 4, the measured air pollution and weather datasets presented in this chapter will be combined with simulated data generated by a chemical transport model. The readings obtained from the monitoring network were also used as a benchmark to evaluate performance of the air quality modelling system. In Chapters 5 and 6, the health, observed air pollution and weather data presented in this chapter as well as the simulated air pollution and weather data presented in the next chapter will be used to estimate relative health risks of air pollution by applying a time-series approach.

Chapter 4 The use of air quality modelling and a blending technique in generating air pollution and weather data

4.1 Introduction

In the previous chapter, the weather and air pollution data described were based on measurements from the weather and ambient air quality monitoring networks. There is another set of weather and air quality data used in Component 1 of this thesis, as set out in Chapter 1, for the purpose of constructing epidemiological models to estimate relative risks of acute health outcomes associated with simultaneous exposure to air pollution and temperature. Thus, this chapter serves the same purpose as the previous chapter, but focuses on the second set of weather and air pollution data.

This chapter begins by discussing the rationale for using this dataset. It has its basis in the review of the roles of air quality modelling in Chapter 2 and adds a rationale for the blending of observed with modelled data. Then, methods employed to generate this dataset, from running a meteorological and air quality modelling system and further blending the simulated output with observations, are explained. Next, model performance and the extent to which the blended data are an improvement relative to the modelled data are evaluated. Similar to other approaches aiming to improve estimates of weather and air pollution, the methods applied here have advantages and disadvantages, which are discussed. The final section of this chapter outlines how the blended data are applied in the following chapter.

4.2 Rationale for blending observations and simulated weather and air pollution data

In projecting the future health impacts of air pollution, as described for the health impact function of Equation 1.1, air quality and weather data are used to estimate the baseline log relative risks (β) and to predict estimated changes in concentrations of the pollutants of interest between the baseline and future periods (ΔC). The latter are derived from a climate-air quality modelling system and are then included in the function. As introduced in Chapter 1, more reliable future projections could be made if consistent methods were used for these two purposes. Therefore, using pollution concentrations and weather parameters simulated from a climate-air quality modelling system for epidemiological modelling, the same modelling system used to project future climate and air quality, is the starting point of this chapter.

Despite several benefits of using the simulated data generated from air quality models in epidemiological studies of air pollution and health, as reviewed in Chapter 2, air quality models have limitations in their performance. When compared side-by-side to observations obtained from a monitoring station, strong over-predictions or under-predictions can be found, depending on the variables of interest, spatial and temporal scales of concern and chemical mechanisms used to drive the models. For example, evaluation of the performance of CMAQ, a chemical transport model developed by the United States Environmental Protection Agency (USEPA), indicated that there was a limited range of daily O_3 max 8-h concentrations between 35–85 ppb within which the model could accurately predict (Appel et al. 2007). For $PM_{2.5}$, given its nature of multiple constituents, each constituent often varies seasonally due to variations in emissions sources, response to meteorological conditions and chemical reactions in the atmosphere (Appel et al. 2008). As a result, the seasonal patterns found for each component lead to seasonal variation in the performance of the model in predicting total $PM_{2.5}$ mass.

Given the limitations in performance of models to date, an alternative approach is to combine multiple sources of measured air pollution data with modelled data. A range of techniques is available for this purpose. One of the methods is to assimilate surface observations into a gridded model field to correct model biases and improve model predictions. As reported later in Section 4.4.1, the modelled data generated by an air quality modelling system—TAPM-CTM—and used in this chapter contained biases and errors, when compared to observations. Hence a blending technique, which was reported to be capable of correcting the biases and errors in the TAPM-CTM modelled fields (Physick et al. 2006), was used to generate a second set of air pollution and weather data to further estimate the baseline relative risks of acute health outcomes associated with air pollution. Apart from the blending technique used in this chapter, other techniques have been developed and the research in this area is highly active. One recent technique, for example, is to use the Bayesian Maximum Entropy method to combine measured data from monitoring stations with other sources of model outputs including land use regression estimates and model fields simulated from a chemical transport model (Akita et al. 2014; Nazelle et al. 2010).

A comparison between four approaches, as introduced in Chapter 1, that used observations and the blend of modelled and observed data aggregated and estimated at different spatial

levels of the Melbourne Region to estimate the relative risks, is made in Chapter 5 to address Objective 1 of this thesis.

4.3 Methods

There are two steps in generating a blended dataset of observations and simulations. The first step involves running a modelling system to generate weather and air pollution output. This is followed by blending the modelled output with observations obtained from monitoring stations in the study domain. This section explains these two steps.

4.3.1 Meteorological and air quality modelling system

In this thesis, the TAPM-CTM modelling system was used to simulate air pollution and weather data. This is an urban-regional scale atmospheric modelling system developed by the CSIRO. The modelling system consists of two components, TAPM, which is a meteorological component, and CTM, which is a chemical transport component. Although the TAPM component has the air quality predictions functionality, similar to the CTM component, the predictions of air quality presented in this chapter do not rely on the TAPM component. Rather, the CTM component, which has more advanced chemical mechanisms than those developed for the TAPM component, was used to estimate air pollution concentrations.

The meteorological component of TAPM basically uses a set of databases—including terrain, vegetation, soil types, leaf area index and synoptic scale meteorology—to simulate meteorological conditions. The meteorological conditions predicted are used to drive atmospheric chemistry in the CTM component and to predict air pollution concentrations. In the CTM component, an emissions inventory is needed to provide estimated emissions of air pollution from anthropogenic and natural sources. More details of the TAPM-CTM are provided in its technical papers and user manuals (Hurley 2008a; Hurley 2008b; Cope and Lee 2009b, a).

Model characteristics described in this section are specific for weather and air quality simulations during the baseline period 1999 to 2008. Although the same modelling system was used to simulate future climate and air quality for the periods 2025 to 2034 and 2065 to 2074, some model characteristics were different. The modelling system and its characteristics for the future projections are elaborated later in Chapter 8.

The model characteristics used to simulate the present-day weather and air quality were as follows.

4.3.1.1 Spatial scale

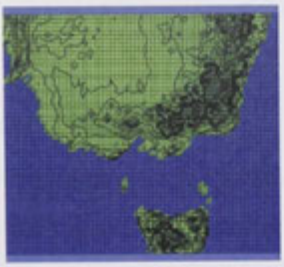
TAPM version 4.0 was the modelling system used to simulate weather and air pollution fields in this chapter. The modelling system was configured with three nested domains with grid sizes from the inner to outer domains of 3, 9 and 18 km respectively. Each domain had 70*60 grid fields in the horizontal and 25 levels in the vertical. The modelling system was centred on Melbourne at the coordinate -37.82°S, 144.97°E (see Figure 4.1).

The CTM component was configured in a consistent fashion to TAPM. The CTM system consisted of three domains with grid sizes and the centre of the domains identical to the TAPM component. The number of grid fields in each domain was lower than TAPM; in the horizontal 50*50 and in the vertical 16 levels reaching to a height of 5 000 metres.

Only the simulations of the first vertical layer of TAPM and CTM inner grid domains (4 200 grid cells for the TAPM component and 2 500 grid cells for the CTM component) were subsequently used to blend with observations. The size of the inner grid domains was sufficiently large to cover the Melbourne Region.

4.3.1.2 Emissions inventory

The air emissions inventory supplied to the modelling system was the information of estimated emissions in Victoria during 2006 developed by EPA Victoria (EPA Victoria and CSIRO 2007). The inventory contained information on a number of pollutants including CO, NO_x, NH_3, PM, SO_2, VOCs and hazardous air pollutants emitted from both anthropogenic and biogenic sources. While the spatial coverage for this inventory was the entire Victorian region, greater details of the emissions were provided for the PPR and Latrobe Valley where anthropogenic emissions are more intense and populations are denser.



(a) Outer domain at grid spacing 18 km

(b) Middle domain at grid spacing 9 km

(c) Inner domain at grid spacing 3 km

Note: Blue and green colours represent water and land areas respectively.

Figure 4.1. TAPM grid fields of the three nested grid domains

4.3.1.3 Chemistry for the CTM component

Chemical mechanisms applied in the modelling system were Carbon Bond 2005 and aerosol species (CB05_aert). These mechanisms are suitable to simulate photochemical pollutants, in particular O_3, as well as reactions of the primary and secondary $PM_{2.5}$ from urban sources (Cope and Lee 2009a). $PM_{2.5}$ simulated from CB05_aert consisted of 16 component species including ammonium ions, aerosol nitrate, aerosol sulphate, sea salt, elemental carbon, nine species of secondary organic aerosol, primary organic carbon aerosol and miscellaneous aerosol species. Concentrations estimated for these component species were aggregated to compute total $PM_{2.5}$ mass presented in this chapter. As mentioned in Chapter 1, the pollutants from dust and bushfires episodes were not included in the analyses of this thesis; thus, the options for fire and dust were disabled in this modelling system.

4.3.2 Blending modelled and observed weather and air quality data

The method developed by Physick et al. (2006) was adopted here to blend daily measurements and simulations of weather and air pollution data. The principle of this method is to correct the modelled data at a given grid cell by using a weighted average of the differences between daily observed and modelled data from all the monitoring stations within the network boundary as shown in Equation 4.1.

$$\emptyset_{ij}^{blended} = \emptyset_{ij}^{modelled} + \sum_{k=1}^{N} w_{ijk}(\emptyset_{k}^{observed} - \emptyset_{k}^{modelled}) \; / \sum_{k=1}^{N} w_{ijk} \quad (4.1)$$

In Equation 4.1, N refers to the number of monitoring stations. $\emptyset_{ij}^{blended}$ and $\emptyset_{ij}^{modelled}$ represent blended and modelled data at grid cell ij respectively. $\emptyset_{k}^{observed}$ and $\emptyset_{k}^{modelled}$ represent observed and modelled data at monitoring station k respectively. W_{ijk} is a weighting function of the difference between daily observed and modelled data at monitoring station k and is calculated as depicted in Equation 4.2.

$$W_{ijk} = \left\{\left[1 - \left(\frac{x_{ij}-x_k}{a}\right)^2 - \left(\frac{y_{ij}-y_k}{b}\right)^2\right]^n\right\}^+ \quad (4.2)$$

At a grid cell ij, a weight was computed by taking into account the distance between the grid cell ij and a monitoring station located at point k along x and y axes of a Cartesian coordinate system. The Cartesian distances along the x and y axes were proportional to a and b, lengths of the major (x) and minor (y) axes reflecting spatial scales being optimal to the monitoring network as well as dispersion and distribution of each weather and air pollution variable in the study area. Identifying the optimal length scales (a,b) for each variable was a prior step to computing the weight (see Appendix B.1 for optimal length scales calculated for each variable in each season). n is a shape factor and determines how quickly w_{ijk} approaches zero as x_{ij}-x_k and y_{ij}-y_k approach a and b respectively. Based on Physick et al. (2006), the shape factor of two was identified to be suitable to the monitoring network in Melbourne and that value was applied here. Thus, a and b define an ellipse around each grid-point within which stations are given positive weight.

Sources of the observed data used in the blending method for each weather and pollution variable were the same as previously described in Chapter 3. As outlined in Chapter 1, estimations of baseline relative risks conducted in Component 1 of this thesis were restricted to days in which the air quality in the Melbourne Region was not considerably affected by emissions from bushfires and dust storms. In line with such a restriction, on days in which readings of PM_{10}, $PM_{2.5}$ and O_3 concentrations in any of the stations located in the Melbourne Region exceeded the air quality standards and bushfires were identified as inferred causes (see Appendix A.4a), $PM_{2.5}$ and O_3 data were excluded from the blending processes. Similarly, on days in which readings of PM_{10} and $PM_{2.5}$ concentrations exceeded the air quality standards and dust events were identified as the likely cause (see Appendix A.4b), $PM_{2.5}$ data were not included in this analysis.

4.4 Performance evaluation

This section presents an evaluation of the performance of modelled air pollution and weather data generated from the TAPM-CTM modelling system and of the data after the blending method was applied versus observations obtained from the monitoring networks. The main purpose of the evaluations made in this section is to determine whether or not outputs derived from the modelling system and from applying the blending method are sufficiently reliable to be further applied in epidemiological studies. This type of evaluation can be referred to as 'Operational Evaluation' (Dennis et al. 2010), and involves the use of a set of performance indicators to assess the magnitude of model biases, errors and agreements. Although this section is not aimed at identifying factors underlying model deficiencies, some potential causes of the deviations between the modelled data and the observed data are briefly discussed.

In this chapter, indicators selected for the performance evaluation included mean bias (MB), gross error (GE), fractional bias (FB), fractional error (FE), root mean square error (RMSE), correlation (R) and index of agreement (IOA). These indicators are principally recommended in evaluating performance of atmospheric and chemical transport models (Chang and Hanna 2004; Thunis et al. 2011; Dennis et al. 2010). These selected indicators aimed at measuring four types of performance measures, including: i) bias (MB and FB), ii) error (GE and FE and RMSE), iii) correlation (R), and iv) level of agreement (IOA).

The following section briefly describes what each indicator measures and how to interpret it (see details of calculation of each performance indicator in Appendix B.2). MB is an average of the differences of all the modelled and observed pairs of interest. Positive and negative values of this measure indicate over-predictions and under-predictions respectively. An ideal value of MB is zero. GE is similar to MB except that the absolute value of the difference of each modelled and observed pair is used so that GE is always positive. A lower value of GE indicates better performance of the modelling system evaluated. Similar to GE, RMSE values are always positive because the discrepancy in each modelled and observed pair is squared. The ideal value of RMSE is zero. The unit of FB and FE is percentage. FB ranges from -200% to 200%, while FE is in the range of 0% to 200%. FB and FE are modifications of MB and GE respectively, in that the difference of each modelled-observed pair is normalised by the addition of each modelled-observed value that is divided by two. The methods of calculation of FE and FB make them less

affected by extreme values of modelled and observed data. R and IOA are generally used to gauge the agreement of modelled and/or observed data. The values of R range from -1 to +1; values closer to + 1 indicate better agreement. Negative values of R indicate an inverse relationship between the modelled and observed data. IOA is always positive as its formula contains squared and absolute terms. The ideal value of IOA is 1, indicating perfect agreement between the two datasets.

To evaluate the performance, the modelled and blended data were compared with observed data at grid cells where the monitoring stations are located (13 stations for O_3, 11 stations for $PM_{2.5}$, 23 stations for dry-bulb temperature and nine stations for dew point temperature). To provide an overall picture of how well the modelling system and the blending method perform in estimating pollutant concentrations and weather parameters for the entire domain, daily modelled/blended and observed pairs were averaged across all the monitoring stations before being subsequently used to calculate the indicators. For each variable, apart from considering the model performance over the whole period, the data were seasonally partitioned to make an analysis of inter-seasonal variability across the four seasons.

Due to some technical problems in running the TAPM-CTM models such as instability in winds in the modelling system, the data for January 2000 and for three days (31 July 2000 and 1 to 2 March 2004) could not be simulated and were not included and analysed in this chapter.

Other than the use of a prior set of criteria or goals to benchmark performance of the model of interest, another practice to verify model performance is to make an inter-comparison with other modelling systems. The inter-comparison approach generally requires collaboration from different atmospheric institutions, involvement of numerous personnel and a great deal of time and financial resources. Thus it was impossible for this thesis to conduct it. Nevertheless a few studies have used this approach to assess the performance of TAPM or TAPM-CTM. For example Hurley and Luhar (2005) compared three models—TAPM, AUSPLUME, and CALPUFF— against two international tracer datasets (Kincaid and Indianapolis). In that study, TAPM performance for Kincaid was similar to the other two models, while for Indianapolis TAPM performed better than AUSPLUME and CALPUFF.

In this section, as presented in Chapter 3, the weather and pollution variables included in the performance evaluation are as follows:

- O_3: daily average (O_3 24-h) and three maximum averaging times (O_3 max 1-h, O_3 max 4-h and O_3 max 8-h).
- Combined $PM_{2.5}$: daily average ($PM_{2.5}$ 24-h).
- Dry-bulb temperature: daily average (temp 24-h), daily maximum (temp max 1-h) and daily minimum (temp min 1-h).
- Dew point temperature: daily average (dew 24-h), daily maximum (dew max 1-h) and daily minimum (dew min 1-h).

4.4.1 Model performance before application of the blending method

In this section, the ability of TAPM-CTM to estimate each air pollution and weather variable was mainly evaluated by benchmarking against criteria and goals proposed by past studies for some of the seven indicators (see Table 4.1). The goals are stricter than the criteria. Thus a modelling system that its performance meets the goals would be more desirable. The criteria are set to be the minimum performance that a modelling system must meet. No criteria and goals were found in the literature for dew point, so the only criterion applied here was IOA, as adapted from that of humidity (Emery et al. 2001). Other than evaluating the model performance by using the indicators, a set of box plots was also used to identify some possible causes of model deficiencies.

Tables 4.2 and 4.3 show average concentrations of observed and modelled air pollution and weather data respectively across the entire domain and over the period 1999 to 2008 with the seven indicators for each variable.

Table 4.1 Criteria and goals for some performance indicators obtained from the literature to be used in evaluating the TAPM-CTM performance

Parameter	Indicator	Criterion	Goal
Dry-bulb temperature[a] (°C)	GE	<2	NA
	MB	-0.5<MB<0.5	NA
	IOA	>0.8	NA
Dew point temperature[a]	IOA	>0.6	NA
$PM^{b,c}$	FB	-60%<FB<60%	-30%<FB<30%
	FE	<75%	< 50%
	IOA	>0.6	NA
$O_3^{c,d}$	FB	-30%<FB <30%	-15% <FB < 15%
	FE	<50%	<35%
	IOA	>0.6	NA

Note: [a] proposed by Emery et al. (2001). The original unit for temperature was Kelvin; [b] proposed by Boylan and Russel (2006) for FB and FE; [c] proposed by Juda-Rezler et al. (2012) for IOA; [d] proposed by Morris et al. for FB and FE

4.4.1.1 Ozone

Overall, the model performs moderately well in predicting O_3 concentrations. The FB, FE and IOA values calculated for all four O_3 variables met the criteria levels when the modelled-observed data were analysed for all-seasons. For the O_3 maximum variables, most of the values of FB and FE, which were better than the goals suggested that the model performed exceptionally well in predicting peak concentrations. The model had a tendency towards over-prediction, indicated by positive MBs and FBs for all O_3 variables across all seasons. The FB and FE values computed for O_3 24-h across all seasons were higher than those for O_3 maximum variables, probably indicating that the model was able to predict peak concentrations better than low or background concentrations. Considering the model performance by season, concentrations of the O_3 maximum variables were under-predicted in the spring as suggested by negative biases. The under-prediction was also apparent in the summer restricted to only O_3 max 1-h levels. Higher GE, RMSE and FE values found in the summer compared to the other seasons for O_3 maximum variables suggested that the model did not perform well in predicting upper extreme O_3 levels during O_3 episodes, which usually occur in summer times.

Generally, the observed and modelled O_3 concentrations had moderate agreement, indicated by R values above 0.5 and IOA values above 0.6 across all seasons. When separated by season, poor agreement between the observations and predictions indicated by

low R and IOA values was found mostly in the winter for all O_3 maximum variables. The lowest level of agreement between observed and modelled data was found for O_3 max 1-h in winter ($R \approx 0$). The observed and modelled data for O_3 24-h in the summer also had poor agreement, indicated by low R and IOA values ($R = 0.272$, $IOA = 0.463$).

Model performance across concentration ranges was further explored by box plots to depict the results of the model performance described above. It can be seen from the plots in Figure 4.2 that the model was able to predict O_3 24-h well in the range between the 50th to 90th percentiles (~15–20 ppb); below and above this range, the O_3 24-h concentrations were over-predicted and under-predicted respectively. For maximum O_3 concentrations, the model was able to predict moderately well at a wider range across the 30th to 80th percentiles. The plots suggested that the model performed best when maximum O_3 concentrations were within the range between 20–30 ppb and average O_3 concentrations were within the range between 15–20 ppb. The concentration ranges best predicted by the model provided an explanation for the poor agreement between observed and modelled data in the winter discussed previously for O_3 maximum variables. This occurred because the model poorly predicted maximum O_3 concentrations at levels lower than 20 ppb, which are common in the winter.

One potential cause of the over-predictions when the observed O_3 levels were relatively low, particularly in the winter, might be related to under-predictions of NO_x. Box plots in Figure 4.3 show that NO_2 average 24-h and maximum 1-h concentrations were strongly under-predicted in the winter. Having insufficient quantities of NO_2 in the modelling system in a winter time or at night could lead to an over-prediction of O_3 as a result of lack of NO_x to titrate with surface O_3.

Table 4.2 Performance indicators between observed and TAPM-CTM modelled air pollution data over the period 1999 to 2008 in the Melbourne Region

Air pollution variable	No. of days	Observed mean	Modelled mean	MB	GE	FB (%)	FE (%)	R	RMSE	IOA
O_3 24-h (ppb)										
All-seasons	3571	14.6	17.2	2.6	4.7	23.3	33.6	0.514	5.8	0.611
Autumn	908	12.5	15.9	3.5	5.0	30.8	39.2	0.426	5.9	0.565
Spring	905	17.5	18.0	0.4	5.0	5.8	21.4	0.426	5.9	0.567
Summer	840	16.4	19.2	2.8	5.5	20.7	32.5	0.272	6.5	0.463
Winter	918	12.2	15.9	3.7	4.8	35.3	41.1	0.606	5.8	0.629
O_3 max 1-h (ppb)										
All-seasons	3571	27.2	27.5	0.3	6.0	5.2	22.1	0.604	8.1	0.696
Autumn	908	25.4	27.6	2.2	6.1	12.3	23.5	0.590	7.9	0.679
Spring	905	29.6	28.0	-1.6	6.1	-4.0	15.9	0.590	7.9	0.680
Summer	840	31.6	31.4	-0.1	9.0	7.2	28.7	0.628	11.3	0.659
Winter	918	22.7	23.4	0.7	4.5	5.3	20.7	-0.004^{*ns}	5.9	0.337
O_3 max 4-h (ppb)										
All-seasons	3573	25.5	26.3	0.9	5.9	7.8	23.2	0.582	7.7	0.679
Autumn	908	23.6	26.2	2.6	6.0	14.7	25.2	0.573	7.6	0.657
Spring	907	28.1	26.9	-1.2	6.0	-2.7	16.3	0.573	7.6	0.658
Summer	840	29.3	30.1	0.8	8.5	9.7	29.2	0.598	10.4	0.637
Winter	918	21.2	22.5	1.4	4.6	9.5	22.8	0.068	6.0	0.370
O_3 max 8-h (ppb)										
All-seasons	3573	23.0	24.0	1.0	5.6	9.6	24.9	0.584	7.2	0.661
Autumn	908	20.8	23.3	2.6	5.6	16.9	27.3	0.577	7.0	0.638
Spring	907	26.0	24.7	-1.2	5.6	-2.9	17.2	0.577	7.0	0.639
Summer	840	26.9	27.5	0.7	7.8	9.2	29.2	0.535	9.6	0.584
Winter	918	18.6	20.6	2.0	4.5	15.2	26.2	0.297	5.9	0.464
$PM_{2.5}$ 24-h ($\mu g/m^3$)										
All-seasons	3545	7.0	4.2	-2.8	3.0	-48.3	51.2	0.532	4.3	0.591
Autumn	897	8.1	5.1	-3.0	3.6	-43.7	51.3	0.433	5.2	0.573
Spring	897	5.9	3.6	-2.2	3.6	-47.9	49.0	0.433	5.2	0.573
Summer	836	6.5	4.0	-2.4	2.5	-43.1	44.7	0.404	3.7	0.480
Winter	915	7.7	4.2	-3.5	3.5	-58.1	59.3	0.632	5.1	0.629

*Correlations are all significantly different from zero at $p < 0.05$ except the single value denoted 'ns'

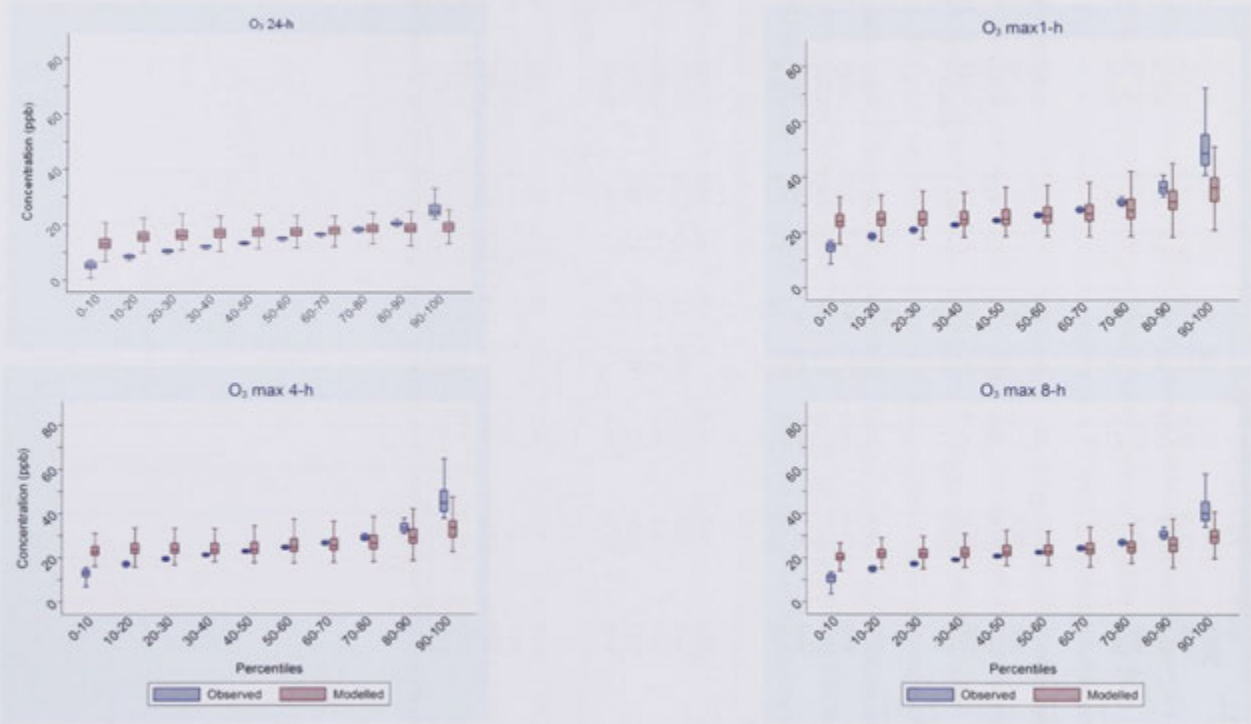


Figure 4.2 Box plots of concentrations of O_3 24-h, O_3 max 1-h, O_3 max 4-h and O_3 max 8-h at every10^{th} percentile bin of observed values, over the period 1999 to 2008 in the Melbourne Region

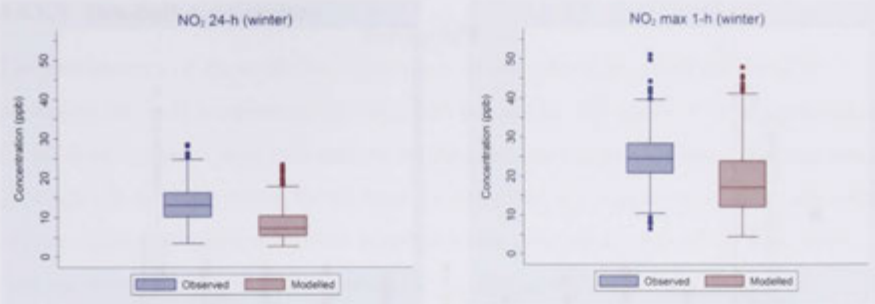


Figure 4.3 Box plots of NO_2 24-h and max 1-h concentrations comparing observed versus modelled data in winters over the period 1999 to 2008 in the Melbourne Region

Another possible factor that could cause the model to predict O_3 concentrations within the limited ranges and be unable to capture concentrations at low and high extremes is the number of vertical layers set up for the TAPM-CTM model runs. In testing CMAQ, a chemical transport model, to identify factors influencing model performance, Appel et al. (2007) found that increasing the number of vertical layers from 14 to 34 could reduce over-prediction of O_3 occurring at levels below 35 ppb and correct an under-prediction occurring at levels above 85 ppb. Such improvement at low concentrations was explained by having less O_3 concentrations aloft penetrating lower layers.

4.4.1.2 PM2.5

Overall, the predictions made by the model for $PM_{2.5}$ 24-h were not as accurate as those for O_3. Nevertheless, the FB, FE and IOA values of $PM_{2.5}$ 24-h for all-seasons suggested that the model still met the criteria levels. The negative MBs and FBs indicated under-predictions over the entire study period and in all-seasons examined. The magnitude of the under-predictions indicated by the MBs is relatively large, approximately 2 $\mu g/m^3$. Nonetheless, there is moderate agreement between the observed and modelled data, indicated by R and IOA being above or around 0.5, except for readings in the summer.

Considering GE, FE and RMSE, the model appeared to predict better in the summer compared to other seasons, although R and IOA in the summer were slightly lower than the other seasons. This might be related to the removal from this evaluation of extreme concentrations of $PM_{2.5}$ caused by bushfires and dust events, a common summer phenomenon.

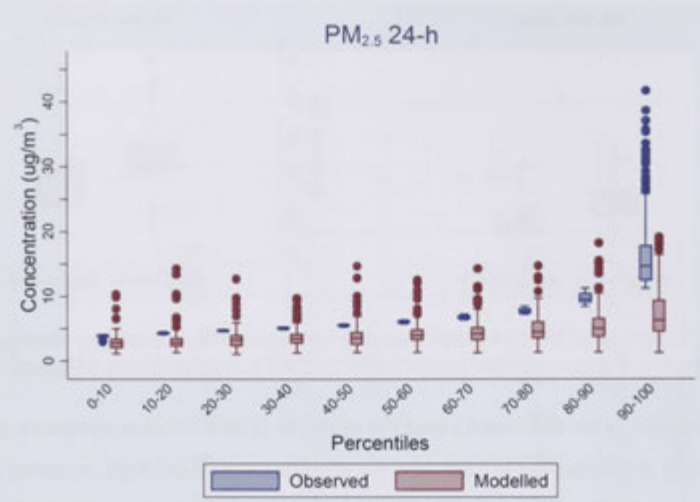


Figure 4.4 Box plots of concentrations of $PM_{2.5}$ at every 10^{th} percentile bin of observed values, over the period 1999 to 2008 in the Melbourne Region

Figure 4.4 suggests that the under-predictions of $PM_{2.5}$ 24-h occured across the entire percentile range, becoming larger at the upper percentiles. Since PM is made up of multiple constituents, it is difficult for this evaluation to determine the causes of the model's apparent deficiency without having observed data for each major PM species. However, there are at least two potential causes of the under-predictions. First, there might have been a number of days with elevated concentrations of $PM_{2.5}$ due to planned burns, bushfire and dust events that were not recorded in the air monitoring reports because the $PM_{2.5}$ emitted from the events was not sufficiently large that the concentrations would exceed the $PM_{2.5}$ standards. As a consequence, the days with such events would have been retained in the observed data used for this evaluation, inflating them and resulting in the under-predictions of the models at the upper-end of $PM_{2.5}$ concentrations. Second, the relatively constant negative bias regardless of the season and the negative bias that proportionally increases over the percentile range suggest that the under-predictions might be, in part, due to having too low background $PM_{2.5}$ concentrations in the modelling system. The low background concentrations may be a result of underestimated emissions from natural sources in the inventory of primary $PM_{2.5}$ or deficiencies of some atmospheric chemical mechanisms to predict secondary $PM_{2.5}$.

4.4.1.3 Dry-bulb temperature

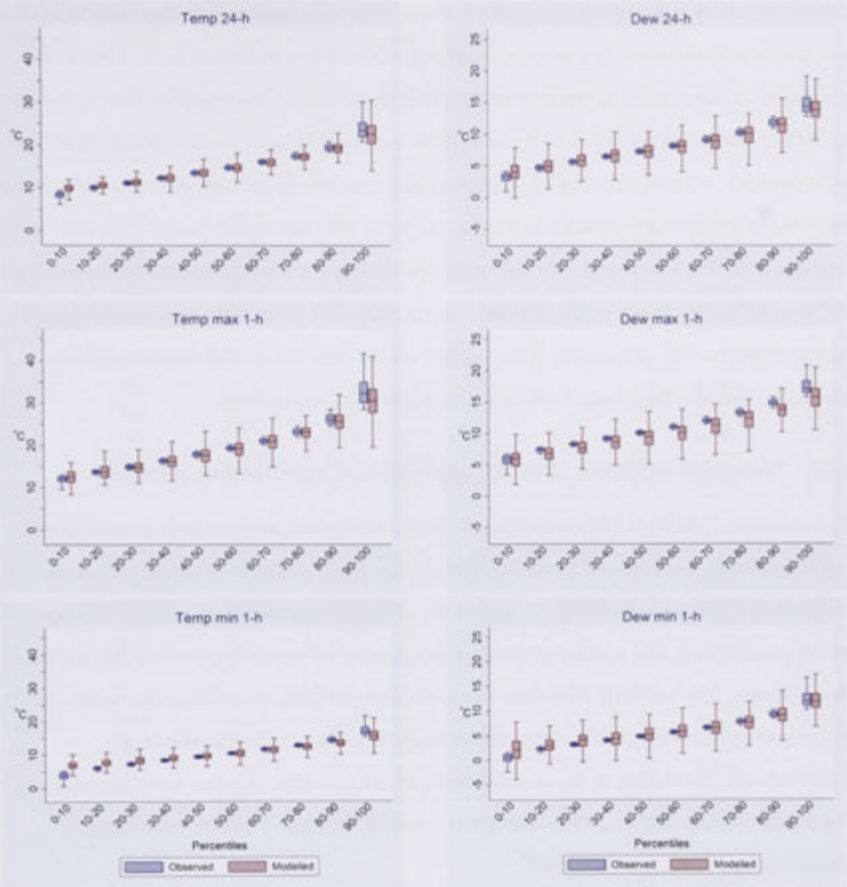
The performance of the modelling system can be considered as a moderate level in predicting dry-bulb temperature, as suggested by the GE, MB and IOA values calculated for all three variables across all seasons meeting the criteria levels. Considering by season, although GE and IOA values for all three variables met the criteria, most of the MB values of these three dry-bulb temperature variables in the summer and the winter were worse than the proposed criteria. Among the three variables, predictions of temp min 1-h appeared to be the poorest, particularly in the winter. This may be due to limited ability of the model to predict low temperatures, as it can be seen from Figure 4.5a that the larger positive biases of temp min 1-h were found at lower temperatures, approximately below the 40th percentiles (~10°C). In the summer, all three temperature variables were slightly under-predicted, particularly temp max 1-h. The under-predictions became noticeable when the maximum temperatures were above 30°C (see Figure 4.5b)

4.4.1.4 Dew point temperature

Using only the IOA indicator, all predictions of the three variables for dew point temperature met the criterion in both all-season and by-season analyses. Dew 24-h and dew max 1-h were slightly under-predicted, while dew min 1-h was slightly over-predicted. In the spring, the slight under-predictions were found for all three dew point variables. Model errors as indicated by GE, FE and RMSE for the three dew point variables were largest in the spring. An agreement between observed and modelled data as indicated by R and IOA was lowest in the winter.

Table 4.3 Performance indicators between observed and TAPM-CTM modelled weather data over the period 1999 to 2008 in the Melbourne Region

Weather variable (°C)	No. of days	Observed mean	Modelled mean	MB	GE	FB (%)	FE (%)	R	RMSE	IOA
Temp 24-h										
All-seasons	3619	14.8	14.8	0.1	1.2	1.5	8.7	0.916	1.9	0.954
Autumn	918	15.2	15.2	-0.1	1.2	0.2	8.6	0.861	1.9	0.923
Spring	910	14.4	14.7	0.3	1.3	2.4	8.9	0.831	2.0	0.909
Summer	872	19.3	18.8	-0.5	1.3	-2.8	6.6	0.868	2.0	0.926
Winter	919	10.3	10.9	0.6	1.1	6.2	10.8	0.747	1.5	0.820
Temp max 1-h										
All-seasons	3619	19.8	19.4	-0.3	1.8	-1.4	9.4	0.903	2.7	0.948
Autumn	918	20.3	19.8	-0.5	1.7	-2.4	8.3	0.886	2.4	0.936
Spring	910	19.5	19.6	0.0	2.2	-0.5	11.3	0.807	3.1	0.896
Summer	872	25.4	24.4	-1.0	2.2	-4.3	9.1	0.860	3.2	0.920
Winter	919	14.1	14.3	0.2	1.3	1.5	9.2	0.727	1.7	0.850
Temp min 1-h										
All-seasons	3619	10.1	10.5	0.4	1.6	8.7	18.5	0.848	2.2	0.899
Autumn	918	10.6	11.1	0.5	1.7	7.0	18.4	0.739	2.3	0.836
Spring	910	9.3	9.9	0.57	1.5	8.5	17.1	0.745	2.2	0.846
Summer	872	13.9	13.3	-0.7	1.3	-4.6	9.4	0.821	1.8	0.889
Winter	919	6.8	8.1	1.3	1.8	23.2	28.8	0.662	2.3	0.715
Dew 24-h										
All-seasons	3611	8.1	8.0	-0.2	1.4	-2.8	20.7	0.840	1.9	0.914
Autumn	918	8.9	9.0	0.1	1.3	2.7	16.3	0.791	1.7	0.888
Spring	910	7.2	6.5	-0.7	1.6	-10.5	28.7	0.719	2.3	0.836
Summer	865	11.0	10.9	-0.1	1.4	-0.3	13.2	0.792	2.0	0.889
Winter	918	5.7	5.6	-0.1	1.2	-2.9	24.4	0.654	1.5	0.808
Dew max 1-h										
All-seasons	3611	10.9	10.0	-1.0	1.5	-10.4	16.5	0.846	2.1	0.900
Autumn	918	11.7	11.0	-0.7	1.4	-6.2	12.6	0.771	2.0	0.864
Spring	910	10.2	8.7	-1.5	1.9	-16.9	22.8	0.716	2.6	0.793
Summer	865	14.1	13.2	-1.0	1.6	-7.0	11.7	0.776	2.2	0.854
Winter	918	7.9	7.1	-0.8	1.3	-11.3	18.8	0.599	1.8	0.738
Dew min 1-h										
All-seasons	3611	5.6	5.9	0.3	1.7	-8.2	72.9	0.780	2.3	0.880
Autumn	918	6.5	7.0	0.6	1.6	7.0	33.0	0.753	2.0	0.856
Spring	910	4.5	4.3	-0.2	2.0	-45.3	140.4	0.642	2.7	0.801
Summer	865	8.1	8.6	0.6	1.8	1.2	38.0	0.760	2.6	0.864
Winter	918	3.6	3.9	0.4	1.5	4.5	78.9	0.608	1.9	0.774



(a) (b)

Figure 4.5 Box plots of weather variables—(a) left panel for dry-blub temperature and (b) right panel for dew point temperature—at every 10th percentile bin of observed values, over the period 1999 to 2008 in the Melbourne Region

Broken down by percentiles, the pattern of under-prediction for low dry-bulb temperatures was also manifested for dew point temperatures as shown in Figure 4.5b. The under-prediction of dew min 1-h became noticeable below the 40th percentiles (5°C), the same percentiles as for temp min 1-h. Results from a comparison of observed and modelled data for temp max 1-h and dew max 1-h across the percentile range suggested that the model was able to predict dry-bulb temperatures at the upper percentiles better than dew point temperatures. For temp max 1-h, the under-predictions were apparent only between the 90th to 100th percentiles, while the under-predictions for dew max 1-h were detectable at greater than the 50th percentile. This might indicate that the model was not able to adequately handle the predictions of water vapour on warm days.

4.4.2 Model performance after the application of the blending method

As described, TAPM-CTM modelling system has limited performance in predicting extreme values, particularly for the air pollutants. Thus using the blending method will lower model biases and errors. To assess the ability of the blending method in improving model predictions, this section presents comparisons of partially blended data versus observations. The partially blended data were derived from correcting the modelled data with observations of neighbouring monitors in the network. For example, O_3 concentrations modelled at the gridded location of a certain monitor were blended with observations from only 12 monitoring sites, out of the total 13 sites, omitting the observations of that monitor itself.

The identical set of indicators was used again to compare the observed versus partially blended data as shown in Tables 4.4 and 4.5 for air pollution and weather variables respectively (see Appendices B.3 and B.4 for comparing observed versus fully blended data). Through the blending method, the model performance was markedly improved. Model biases that were largely due to poor predictions of extreme air pollution and weather levels were lowered for all variables. The negative biases exceeding 2 μg/m^3 for $PM_{2.5}$ 24-h found earlier when comparing the observed with modelled data became less than 2 μg/m^3. Model errors, indicated by GE, FE and RMSE, were reduced approximately by a factor of two. The magnitude of agreement between observed and modelled data was enhanced for all variables, with the largest improved correlation found for O_3 max 1-h in the winter (before the blending R=-0.004; after the blending R=0.941).

Table 4.4 Performance indicators between observed and partially blended air pollution data over the period 1999 to 2008 in the Melbourne Region

Air pollution variable	No. of days	Observed mean	Blended mean	MB	GE	FB (%)	FE (%)	R	RMSE	IOA
O_3 24-h (ppb)										
All-seasons	3569	14.6	15.8	1.19	1.9	13.1	16.2	0.964	2.4	0.949
Autumn	907	12.4	14.0	1.57	2.0	17.1	19.2	0.950	2.4	0.931
Spring	905	17.5	17.9	0.36	1.5	4.6	9.6	0.959	2.0	0.947
Summer	840	16.4	17.7	1.31	2.1	11.3	14.3	0.964	2.6	0.941
Winter	917	12.2	13.7	1.52	1.9	19.3	21.5	0.968	2.4	0.937
O_3 max 1-h (ppb)										
All-seasons	3569	27.2	27.3	0.08	2.1	2.7	8.4	0.973	3.0	0.972
Autumn	907	25.4	26.1	0.69	2.0	5.1	8.7	0.975	2.8	0.973
Spring	905	29.6	29.0	-0.53	1.7	-0.8	5.7	0.964	2.5	0.966
Summer	840	31.6	31.5	-0.08	3.2	3.9	10.9	0.979	4.3	0.970
Winter	917	22.7	22.9	0.22	1.6	2.9	8.3	0.941	2.2	0.933
O_3 max 4-h (ppb)										
All-seasons	3571	25.5	25.7	0.29	2.0	3.8	8.9	0.973	2.8	0.972
Autumn	907	23.6	24.4	0.80	2.0	6.0	9.4	0.975	2.6	0.972
Spring	907	28.1	27.7	-0.35	1.7	-0.2	5.9	0.965	2.3	0.966
Summer	840	29.3	29.6	0.25	3.0	4.8	11.0	0.979	3.9	0.970
Winter	917	21.2	21.6	0.44	1.6	4.7	9.4	0.955	2.3	0.940
O_3 max 8-h (ppb)										
All-seasons	3571	23.0	23.3	0.38	1.9	4.9	9.7	0.975	2.6	0.971
Autumn	907	20.7	21.6	0.89	1.9	7.3	10.6	0.976	2.4	0.970
Spring	907	26.0	25.6	-0.33	1.6	0.0	6.3	0.964	2.2	0.961
Summer	840	26.9	27.1	0.25	2.7	4.7	10.8	0.978	3.5	0.967
Winter	917	18.6	19.3	0.71	1.6	7.5	11.4	0.972	2.2	0.949
$PM_{2.5}$ 24-h ($\mu g/m^3$)										
All-seasons	3543	7.0	5.4	-1.62	1.6	-24.3	24.8	0.959	2.2	0.888
Autumn	896	8.1	6.2	-1.90	2.0	-24.0	25.4	0.952	2.7	0.880
Spring	897	5.9	4.6	-1.26	1.3	-23.6	23.7	0.930	1.5	0.830
Summer	836	6.5	5.1	-1.39	1.4	-22.3	22.7	0.957	1.9	0.867
Winter	914	7.6	5.8	-1.89	1.9	-27.0	27.3	0.968	2.6	0.902

Table 4.5 Performance indicators between observed and partially blended weather data over the period 1999 to 2008 in the Melbourne Region

Weather variable (°C)	No. of days	Observed mean	Blended mean	MB	GE	FB (%)	FE (%)	R	RMSE	IOA
Temp 24-h										
All-seasons	3615	14.7	14.8	0.1	0.3	0.9	2.1	0.996	0.4	0.998
Autumn	917	15.2	15.3	0.0	0.3	0.5	2.0	0.994	0.4	0.997
Spring	908	14.4	14.5	0.1	0.3	1.1	2.2	0.993	0.4	0.996
Summer	872	19.3	19.3	0.0	0.3	-0.1	1.5	0.992	0.5	0.996
Winter	918	10.3	10.5	0.2	0.3	2.0	2.8	0.992	0.3	0.992
Temp max1-h										
All-seasons	3615	19.8	19.7	-0.1	0.5	-0.1	2.3	0.995	0.7	0.997
Autumn	917	20.3	20.2	-0.1	0.4	-0.3	1.9	0.995	0.6	0.997
Spring	908	19.5	19.5	0.0	0.6	-0.1	3.0	0.985	0.8	0.992
Summer	872	25.4	25.1	-0.2	0.6	-0.9	2.2	0.992	0.8	0.995
Winter	918	14.1	14.2	0.1	0.3	0.8	2.1	0.985	0.4	0.992
Temp min 1-h										
All-seasons	3615	10.1	10.3	0.2	0.4	4.0	5.6	0.994	0.5	0.995
Autumn	917	10.6	10.8	0.2	0.4	3.1	4.9	0.991	0.6	0.992
Spring	908	9.3	9.5	0.2	0.4	3.4	4.8	0.991	0.5	0.993
Summer	872	13.9	13.9	-0.1	0.3	-0.2	2.5	0.998	0.5	0.993
Winter	918	6.8	7.2	0.4	0.5	9.4	10.2	0.991	0.6	0.985
Dew 24-h										
All-seasons	3609	8.1	8.4	0.2	0.3	4.7	6.6	0.996	0.4	0.997
Autumn	917	8.9	9.2	0.3	0.3	4.4	4.6	0.996	0.4	0.994
Spring	910	7.2	7.3	0.1	0.3	5.1	11.1	0.993	0.4	0.995
Summer	865	11.0	11.2	0.2	0.3	2.5	2.9	0.997	0.4	0.997
Winter	917	5.7	5.9	0.2	0.3	6.6	7.8	0.988	0.4	0.989
Dew max 1-h										
All-seasons	3609	10.9	10.9	0.0	0.2	0.0	2.0	0.997	0.3	0.998
Autumn	917	11.7	11.7	0.0	0.2	0.1	2.0	0.995	0.3	0.997
Spring	910	10.2	10.1	0.0	0.2	-0.2	2.4	0.994	0.3	0.997
Summer	865	14.1	14.1	0.0	0.2	-0.2	1.2	0.997	0.2	0.998
Winter	917	7.9	8.0	0.0	0.2	0.5	2.2	0.991	0.2	0.995
Dew min 1-h										
All-seasons	3609	5.6	5.8	0.1	0.3	-16.0	32.2	0.995	0.4	0.997
Autumn	917	6.4	6.7	0.2	0.3	-44.6	56.3	0.994	0.4	0.995
Spring	910	4.5	4.6	0.0	0.3	-14.8	30.2	0.993	0.4	0.996
Summer	865	8.1	8.2	0.1	0.3	1.0	5.5	0.996	0.4	0.998
Winter	917	3.6	3.7	0.2	0.3	-4.8	35.2	0.988	0.4	0.993

To explore further the efficiency of the blending method in correcting model biases, scatter plots of $PM_{2.5}$ 24-h, as an example, shown in Figure 4.6, can be used to examine the degree to which the application of the weighting function can help improve model predictions. Before being corrected by the observations, the model performed moderately well, with under-predictions and a moderate level of agreement between observed and modelled data as indicated by an R of 0.532 (see Figure 4.6a). Figure 4.6b suggests that when only observations from neighbouring monitoring stations in the network were used in the weighting function to correct the biases, the agreement between observed and the partially corrected data was dramatically increased. Further, when observations from all the monitoring stations were used to correct the modelled biases, the correlation moved even closer to the stage of perfect agreement (R=0.993).

As previously described, the TAPM-CTM model has the ability to predict air pollution and weather variables within limited ranges. Poor prediction of lower and upper extreme values was one of its weaknesses, in particular for the air pollution variables explored in this chapter. Application of the blending method largely resolved this weakness of the model. It can be seen in Figure 4.7 that most of the outliers at the upper-end of $PM_{2.5}$ 24-h concentrations (the bin of 90^{th} to 100^{th} percentiles) that could not be captured by the model were corrected when the weighting function was applied, both for partially and fully corrected data. Likewise, the outliers of modelled data found at most of the percentile range were noticeably reduced when being corrected by the observations.

Spatial plots of $PM_{2.5}$ 24-h as shown in Figure 4.8a demonstrate how the weighting function operated to correct modelled data in the study domain. Since the model predominantly under-predicted $PM_{2.5}$ 24-h concentrations, the concentrations in grid cells that were under-predicted in the left plot were corrected and became higher in the right plot. This can be seen from having larger areas where the colours indicate higher concentrations in the right plot of Figure 4.8a. For O_3, the corrections made by the weighting function were a mix of fixing the over-predictions at low concentrations and under-predictions at high concentrations (see Note: Black dots in the plots represent locations of monitoring stations where the observed data were blended with the modelled data.

Figure 4.8b). The over-predicted O_3 max 1-h concentrations at grid cells located in the middle (approximately at longitude of 144.9°E to 145.1°E and latitude of -37.9°S to -

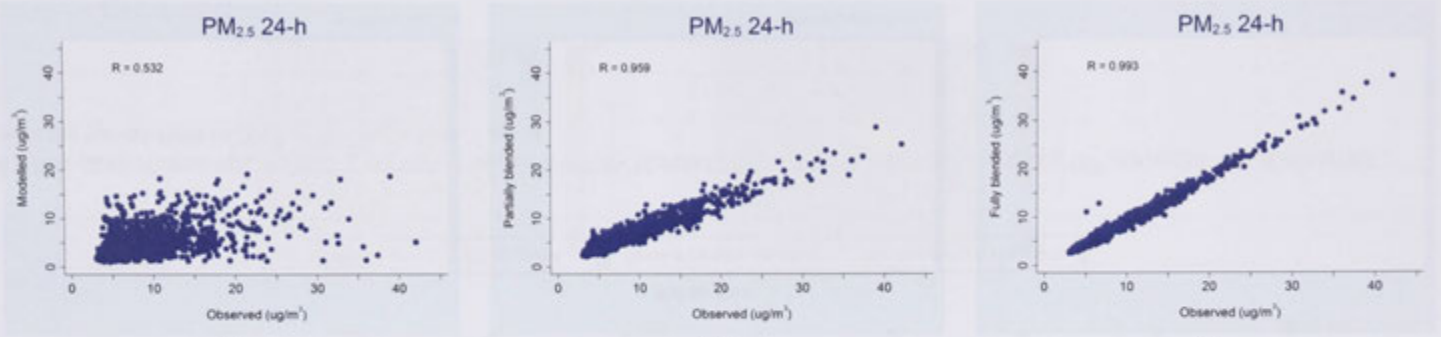
37.7°S) of the left plot of Figure 4.8b—where O_3 precursors were emitted and O_3 were yet been formed—were corrected and became lower in the right plot of Figure 4.8b. On the other hand, grid cells having relatively high O_3 concentrations in the left plot of Figure 4.8b, for example, at the upper north of the domain (latitude of -37.6°S to -37.3°S), were corrected and their concentrations became higher. This can be noticed by expanding the areas where colours indicate higher concentrations in the right plot of Figure 4.8b. It is clear that the corrections mainly occurred within the boundary of the monitoring network as represented by black dots in Figure 4.8.

4.5 Strengths and limitations of the blended data

In combination with the modelled data, the blending method generates output at a finer scale of spatial exposure fields with better accuracy than those available from a monitoring network or an air quality modelling system alone. This is particularly useful for studies investigating health effects as a consequence of variability in exposure to weather and air pollution at the intra-urban level. It is clear based on the evaluation of model performance conducted in this chapter that when observations were incorporated by the blending method into the modelled fields, prediction biases were largely corrected, particularly at both extreme ends of the scale. This is very important in epidemiological studies because adverse health effects tend to be stronger at extreme levels of exposure to air pollution and temperature.

Apart from correcting model biases caused by deficiencies in key model characteristics for example meteorology, boundary conditions and chemical mechanisms, the blending method can improve the modelled data in terms of fixing model biases caused by missing inventory data, in particular for studies that simulate a dataset for a long period.

Generally, collecting data to construct an air pollution emissions inventory—a key component in an air quality model—occurs over a regular period, for example, every five years in Australia. Having emissions inventory data available every year is not possible as producing such data is a time-consuming and expensive process. In practice, an air quality modelling system relies on an emissions inventory for a given year. However, these data may be applied over a simulating time period, which in some cases is longer than a year. This may lead to over-predictions and under-predictions due to variability of air pollution emissions over time.



(a) (b) (c)

Figure 4.6 Scatter plots of observed $PM_{2.5}$ 24-h concentrations across all the monitoring sites in the Melbourne Region over the period 1999 to 2008 against (a) modelled data without any corrections, (b) partially blended TAPM-CTM data by using only neighbouring monitoring stations in the network and (c) fully blended TAPM-CTM data by using all the monitoring stations in the network

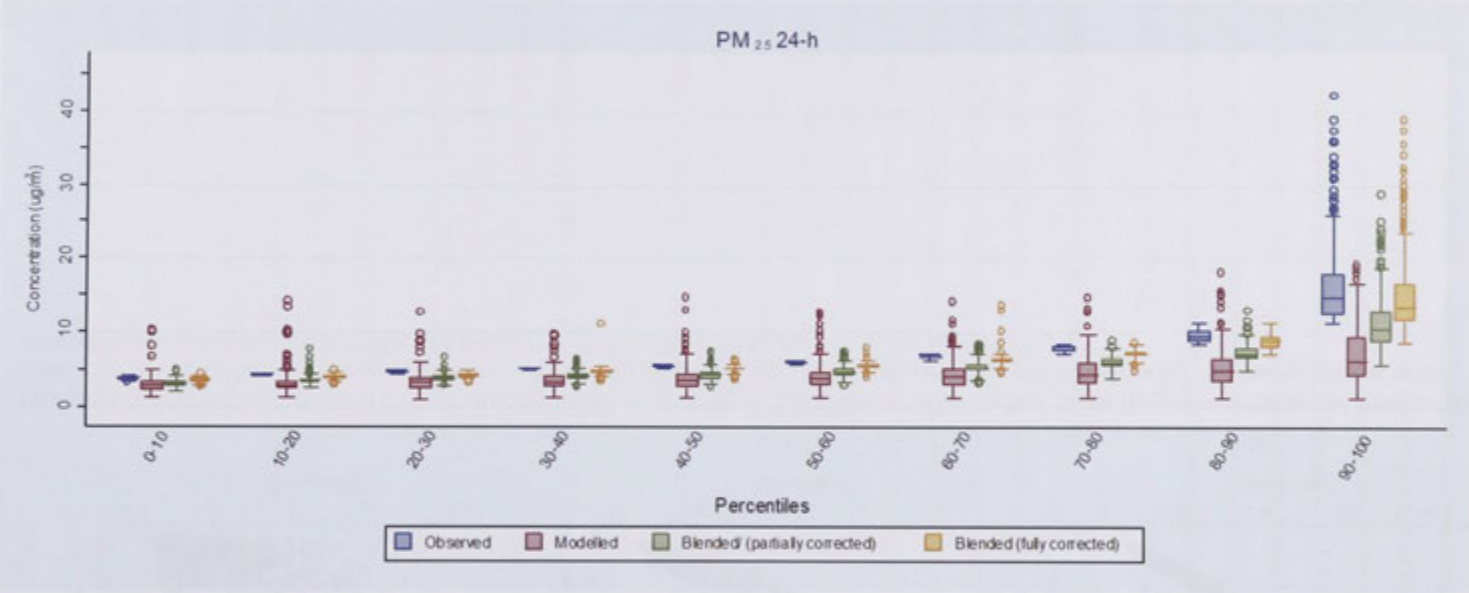
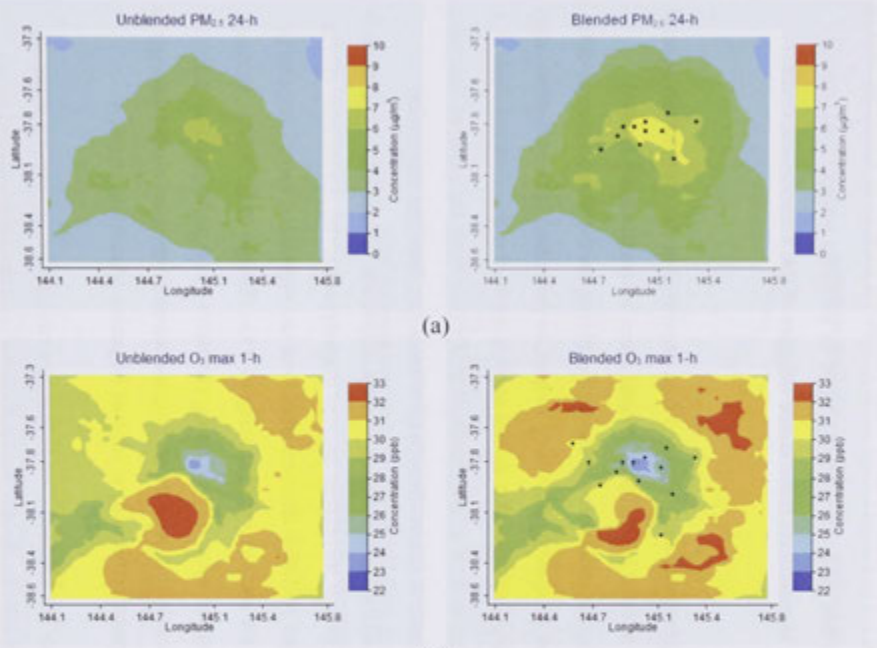


Figure 4.7 Box plots of observed, modelled, partially blended and fully blended $PM_{2.5}$ 24-h concentrations at every 10^{th} percentile bin of observed values, over the period 1999 to 2008 in the Melbourne Region



Note: Black dots in the plots represent locations of monitoring stations where the observed data were blended with the modelled data.

Figure 4.8 Spatial plots of $PM_{2.5}$ 24-h (upper panel) and O_3 max 1-h (lower panel) concentrations comparing between unblended (left) and blended (right) TAPM-CTM fields over the period 1999 to 2008 in the Melbourne domain.

For example, the simulations of the air quality modelling system presented in this chapter were generated based on the 2006 emissions inventory. However, the inventory was assumed to be constant over the period 1999 to 2008. As shown in Figure 4.9, downward trends for CO and NO_2 have been observed from the PPR monitoring network over the past decade. Hence, using the 2006 emissions inventory will have an impact on the accuracy of the model predictions, in particular for the period before 2006 when some actual concentrations for the emitted pollutants were higher than the levels in 2006. In addition, predictions made for pollutants from some emissions sources in which the annual variation is high, e.g. fires and dust, are not as accurate as for those sources that change gradually over time, such as emissions from motor vehicles (EPA Victoria and CSIRO 2007).

Although the blending of observed and modelled data presented in this chapter has several advantages, there are also several limitations. In rural areas where there are limited monitoring sites, estimating air pollution exposures by relying on the two sources of data—monitor measurements and air quality modelling output, as described in this chapter—may be inferior to other blending strategies that do not exploit monitor measurements such as a combination of satellite data and air quality modelling. To overcome the issue of limited number of fixed monitors over a sparse domain of urban areas, land use regression modelling can be used as an alternative source of data to estimate air pollution exposure in the city. This method has gained increasing interest from the scientific community (Hoek et al. 2008). Estimates of air pollution derived from land use regression modelling can be derived at a fine spatial scale such as a grid spacing of 1 km (Briggs et al. 2005). Akita et al. (2014) have reported on the value of combining estimates of air pollution data derived from land use regression modelling with those from a chemical transport model through an advanced geospatial interpolation technique such as Bayesian Maximum Entropy.

Another major limitation of the blended data is that generating them involves a series of long and complex processes. In particular, simulating the modelled data prior to being blended with observations requires considerable computational resources. Logically, as the blending method involves using data generated from an air quality model that has complex functions to correct biases in the modelled data, the blended data should provide better estimates of personal exposure to ambient pollution concentrations compared to other basic approaches, for example, proximity or interpolation methods. However, results from a study that compared blended data generated by the same method used in this chapter with

concentrations obtained from the nearest central monitoring station to estimate ambient outdoor NO_2 concentrations showed that this was not the case (Physick et al. 2011). That study found that estimating ambient concentrations of the pollutant from the nearest station, for the purpose of estimating personal exposure in microenvironments, provided approximations slightly closer to a reference method—personal passive samplers—than the blended data. As long as time and computational resources are of concern, therefore, using a simpler technique like the nearest monitor approach may be more desirable.

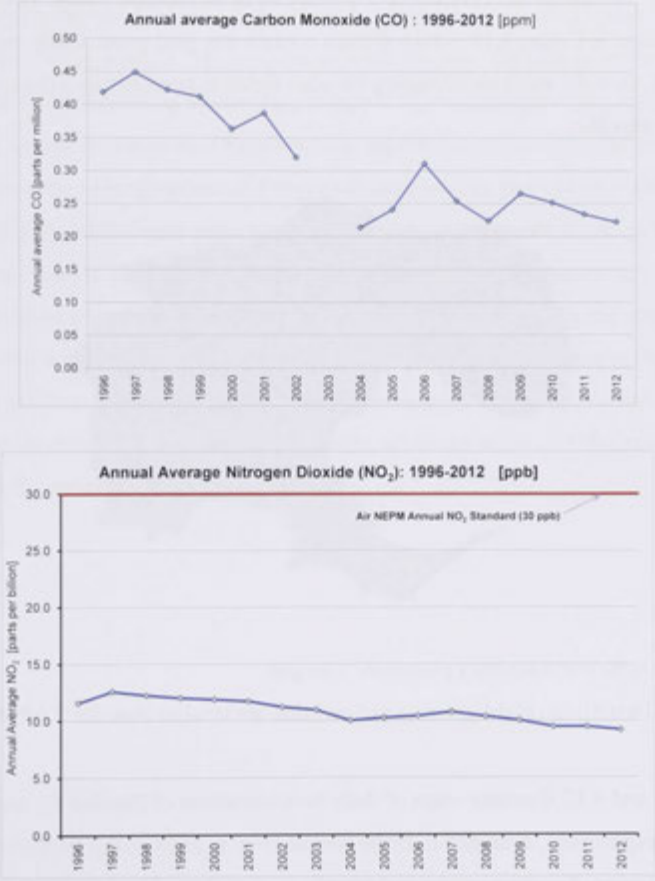


Figure 4.9 Trends over the past decade of CO and NO_2 based on annual averages of observations derived from the monitors in the PPR, 1996 to 2012

Source: EPA Victoria and CSIRO 2013

4.6 Application of the blended data in air pollution-health impact models

To apply the blended fields further in estimating relative health risks associated with air pollution, the fields were aggregated to provide daily weather and air pollution estimates for each SLA in the Melbourne Region. The aggregation for a given SLA was made by averaging all blended points of the air pollution and weather variables identified to be inside that corresponding SLA. For some small SLAs, where there was only one blended point identified to be inside, values from the single blended fields were simply assigned to those SLAs. The largest SLA contained a maximum of 80 blended fields. There were two SLAs, as shown in Figure 4.10, which did not contain any grid point. Daily estimates of these two SLAs were made by averaging blended fields located within a distance of 3 km from their centroids.

Note: Each dot in the map represents a point on the 3-km grid.

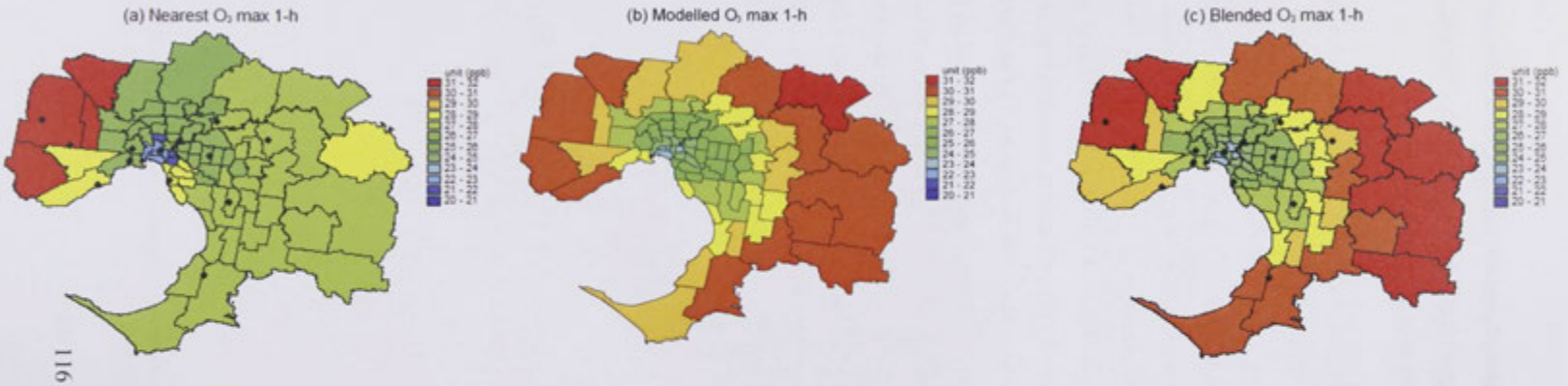
Figure 4.10 Two SLAs (highlighted in yellow) that are smaller than the TAPM-CTM grid size

Figures 4.11 and 4.12 illustrate maps of daily concentrations of blended O_3 max 1-h and $PM_{2.5}$ 24-h respectively, averaged over the period 1999 to 2008 in the 79 SLAs of the Melbourne Region. For the purpose of comparison, the maps also show modelled concentrations by using the same method as described above for the blended data and measured concentrations by estimating from the nearest monitor in each SLA. The map of measured concentrations estimated by the nearest monitors is presented here because this dataset of air pollution and weather will be used to estimate relative health risks of air pollution in Chapter 5. It can be seen that concentration gradients across the study area are

clearly different between the data based solely on measurements and solely on simulations for both pollutants. The concentration gradients from blended fields generated from the blending method are in between the measured and modelled data.

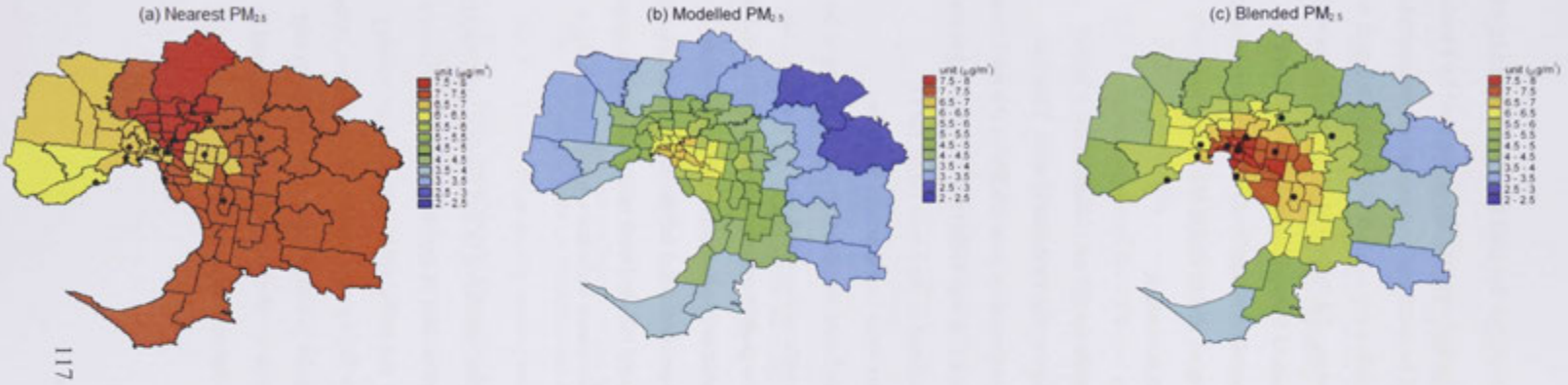
For the pollution data generated by relying on the nearest monitors as shown in Figures 4.11a and 4.12a, there are a number of SLAs, mostly outer SLAs,[1] where their estimated concentrations were highly influenced by only one or two monitors. For the pollution data generated by relying on the simulations as shown in Figures 4.11b and 4.12b, over-predictions and under-predictions compared to those based on the observations from nearest monitors were marked. The concentrations of O_3 max 1-h estimated for outer SLAs by the TAPM-CTM modelling system (see Figure 4.11b) were higher than those estimated based on the nearest monitors (see Figure 4.11a), particularly around the eastern part of the maps. However, the concentrations of $PM_{2.5}$ 24-h estimated by the TAPM-CTM modelling system (see Figure 4.12b) were generally lower than those estimated based on the nearest monitors (see Figure 4.12a). The gradients of $PM_{2.5}$ 24-h concentrations across the Melbourne Region were higher based on the blended data, compared to the observed data from the nearest monitors (see Figures 4.12c and 4.12a). This may be due to the nature of $PM_{2.5}$, which consists of primary and secondary components. Alternatively, such high concentration gradients might reflect the true intra-urban variation of $PM_{2.5}$ concentrations across the Melbourne Region.

[1] Inner areas of the Melbourne Region, where the population density is high and the central business district is situated, are located around the centre of the maps.



Note: Black dots in the plots represent locations of the monitoring network of O_3.

Figure 4.11 Maps of averaged concentrations of O_3 max 1-h over the period 1999 to 2008 at the SLA level in the Melbourne Region based on (a) assigning observed concentrations from the nearest monitor, (b) averaging modelled fields identified to be inside each SLA and (c) averaging blended fields identified to be inside each SLA, as shown in Figure 4.10.



Note: Black dots in the plots represent locations of the monitoring network of $PM_{2.5}$.

Figure 4.12 Maps of averaged concentrations of $PM_{2.5}$ 24-h over the period 1999 to 2008 at the SLA level in the Melbourne Region based on (a) assigning observed concentrations from the nearest monitor; (b) averaging modelled fields identified to be inside each SLA and (c) averaging blended fields identified to be inside each SLA as shown in Figure 4.10.

4.7 Chapter summary

This chapter presented another set of air pollution and weather data generated by blending simulations and measurements. This dataset will be used and compared with measured data in estimating health risks of air pollution in the following chapter. The modelled data were generated by a chemical transport modelling system, TAPM-CTM. A method proposed by (Physick et al. 2006) that used observations obtained from all the monitors within the network boundary was adopted to correct the modelled fields. A set of indicators was used to evaluate the performance of the modelling system and to assess the extent to which applying the blending method could make an improvement.

Results from calculating the performance indicators for the air pollution and weather variables suggested that the modelling system performed moderately well. This was justified on the basis of meeting all the criteria proposed by past studies of the performance values. Based on evaluating the model performance, it was found that the model generally under-predicted at the upper range and over-predicted at the lower range for O_3 concentrations. For $PM_{2.5}$ 24-h, under-predictions were found across the entire concentration range. The model slightly over-predicted minimum dry-bulb temperature at lower range, particularly in the winter, whereas little under-predictions were found for predicting maximum dry-bulb and maximum dew point temperature at the higher end of temperature percentiles. The model was better able to predict maximum temperature at the upper-end of the temperature range than maximum dew point temperature. After applying the blending method, results of comparing partially blended with observed data suggested that the method improved the values of the performance indicators approximately by a factor of two.

Although the results of the performance evaluation presented in this chapter indicated that the blended dataset was reasonable in terms of reduction of exposure bias and accuracy in capturing the spatial variability, this is probably not sufficient to guarantee its validity when applying it to epidemiological studies. The following chapter will, therefore, present an application of the blended dataset to studying the health effects of air pollution and appraise the extent to which this dataset is superior to other datasets commonly used in estimating relative health risks associated with exposure to air pollution.

Chapter 5 Selection of approaches in estimating the health effects of air pollution

5.1 Introduction

This chapter presents an estimation of relative risks for acute health outcomes associated with exposure to air pollution in the Melbourne Region. The datasets of health, air pollution and weather described earlier in Chapters 3 and 4 were used to estimate a dose-response function of mortality and morbidity attributable to ambient pollution exposure. This chapter aims at addressing Objective 1 of this thesis to choose the best approach for estimating the health risks of the air pollution in order to be able to use these risk estimates to assess the impact of future climate change on the health effects of air pollution.

The remainder of this chapter is divided into two parts, Sections 5.2 and 5.3. Section 5.2 involves a selection process among four approaches used for estimating the health effects of air pollution. This section begins with a brief introduction to the four approaches being compared and criteria in selecting them. This is followed by presenting statistical methods and results of two sequential steps in the selection process. Next, the selected approach is applied to estimate the relative risks of all health outcomes of interest. At the end of this section, the results of estimated health risks are discussed on the basis of each category of health outcome, each type of model fitted and explanations for the different relative risks estimated by each of the four approaches. The selected approach will be used in the next chapter to investigate interaction effects between temperature and air pollution on acute health outcomes.

In Section 5.3, the issue of sources and components of $PM_{2.5}$ and their impact on estimations of health risks, as introduced in Section 1.5.3, is explored in two dimensions. The first dimension considers the inclusion of the data on bushfire and dust storm days and its impact on the relationships between air pollution and acute health outcomes that were estimated in Section 5.2. The second dimension explores a possibility of a non-linear relationship between $PM_{2.5}$ and the health outcomes of interest. Such a non-linear relationship may result from the difference in components of ambient $PM_{2.5}$ on days with, versus days without, extreme air pollution episodes. It is also possible that the relationship between individual $PM_{2.5}$ components and health could be linear or non-linear, making a non-linear relationship between total $PM_{2.5}$ mass and health.

5.2 Choice of approaches in estimating the relationship between acute health outcomes and air pollution

A flow diagram of the two steps applied to select the best approach for the subsequent analyses in this thesis is depicted in Figure 5.1. In this section, relative risks for acute health outcomes associated with air pollution estimated by using four different approaches were compared. In the first step, the approach referred to as the 'standard approach' in this thesis, which is the most common approach in time-series studies of air pollution and health to estimate air pollution exposures for a given location, was employed. The approach was used to explore and choose health outcomes that had significant associations with the air pollutants of interest, $PM_{2.5}$ and O_3. For the purpose of selecting *the best approach*, focusing on health outcomes with significant associations made it easier to distinguish the difference in performance of the approaches compared. Statistical models of the standard approach and results of the associations identified from this approach are presented in Section 5.2.1.

In the second step, only health outcomes that had strong associations with the air pollutants based on the standard approach were explored further. Another three approaches were applied to estimate relative risks of the chosen health outcomes. These approaches are referred to here as SLA, nearest and blending approaches. The unit of analysis of all these approaches was shifted from the city to SLA levels to allow exploration of intra-urban heterogeneity. The nearest and blending approaches took into account spatial variations of air pollution exposures over the study area, while the SLA approach did not. To compare the performance of these approaches, the magnitude of the relative risks was the key criterion considered. The approach that provided larger relative risks was chosen. The rationale for this criterion and methods of estimating air pollution exposures for these three approaches and their corresponding statistical models are elaborated in Section 5.2.2. The approach chosen was then applied further in this chapter to estimate relative risks for all health outcomes of interest and in the following chapter to examine how interaction between temperature and air pollution may affect these estimations.

In this chapter and in other places in this thesis a p-value of less than 0.05 has been used as the objective criterion of statistical significance, to facilitate model building. It is however important not to interpret the results as indicating that a significant finding necessarily implies that the association is real, or that a non-significant result implies that no

association is present. The results from this thesis add to, and should be considered in the context of, the evidence from previous studies on the health risks of air pollution exposure in Australian cities.

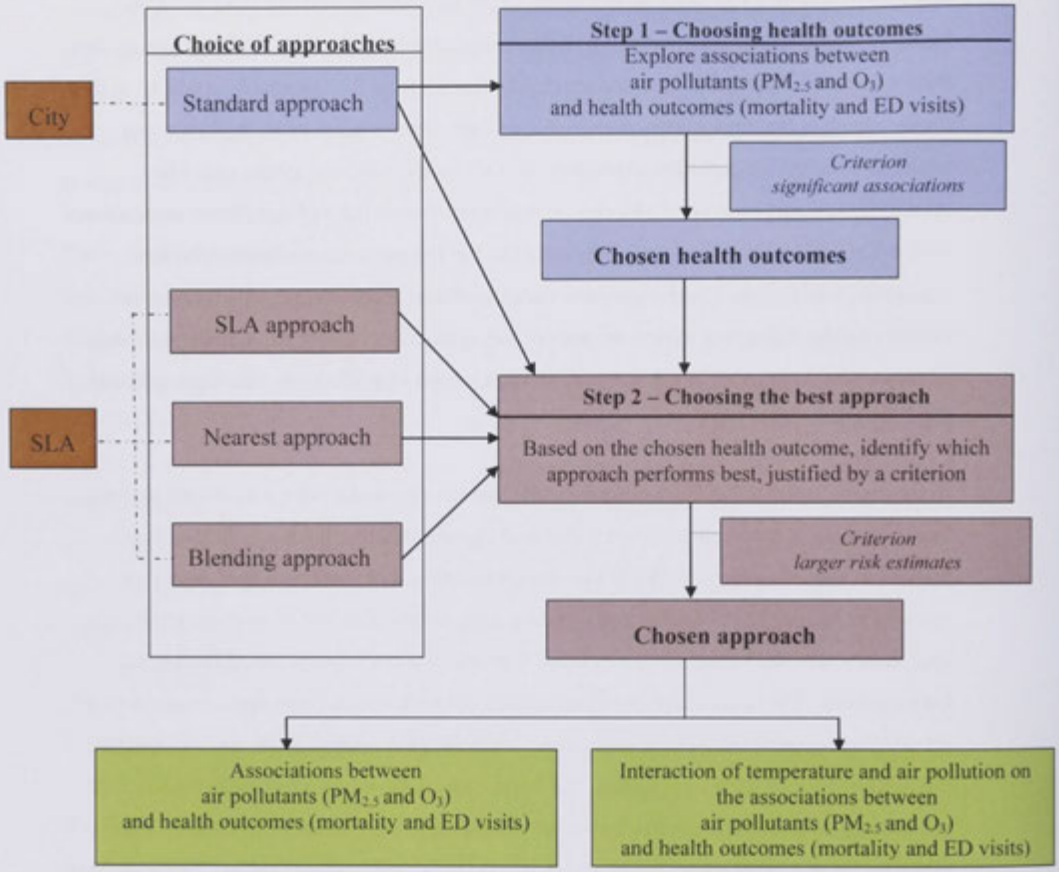


Figure 5.1 Flow diagram of steps in identifying the best approach for further investigating the interaction effects of temperature and air pollution on acute health effects related air pollution

5.2.1 Choice of health outcomes

5.2.1.1 Statistical methods

In this first step, the standard approach was applied to explore the relationships between air pollution and possible health outcomes of interest. The outcomes that had significant associations were selected for further use in the second step. As described in Chapter 3, daily measurements from 14 sites monitoring ambient air quality and 23 sites monitoring weather were averaged to provide a single measure of each air pollution and weather variable for the whole Melbourne Region. The daily mortality and morbidity counts at lower hierarchies of the geographical structure were also aggregated to provide single daily counts for the whole city.

To explore the relationships, the daily estimate of each air pollutant and weather variable was entered into a Poisson regression model of the form:

$$log\,(\mu_t) = \alpha + \beta_1 pollutant_{t,0-2} + s_1(avgtemp_t, 6df) + s_2\big(avgtemp_{t,1-3}, 6df\big) + s_3(avgdew_t, 4df) + s_4\big(avgdew_{t,1-3}, 4df\big) + s_5(time, 7df\ per\ year) + \boldsymbol{\gamma_1 DOW + \gamma_2 influenza + \gamma_3 holiday}, \quad (5.1)$$

where μ_t denotes the observed count of the health outcome of interest on day t; α is the intercept term; β_1 refers to the main effect of air pollution; s_{1-5} refer to smoothing functions with the specified degrees of freedom for average dry-bulb temperature on day t (s_1), average dry-bulb temperature over the previous two days (s_2), average dew point temperature on day t (s_3), average dew point temperature over the previous two days (s_4) and time (s_5); γ_{1-3} are vectors of coefficients that contain the adjustments for the day of the week (*DOW*), influenza epidemics (*influenza*) and holiday (*holiday*) respectively; *pollutant* is the 3-day moving average concentration of the pollutant of interest ($PM_{2.5}$ or O_3); *avgtemp* is the mean of maximum dry-bulb temperature on day t and minimum dry-bulb temperature on day *t-1*; *avgdew* is daily 24-h average dew point temperature; *time* is a day sequence; *DOW* is a set of indicator variables for the day of the week; *influenza* refers to days with and without an epidemic of influenza and *holiday* is an indicator variable of holiday and non-holiday.

In time-series studies of air pollution and health, apart from the air pollutant variable, other variables including dry-bulb and dew point temperatures, calendar time as a proxy for

predictors that have long-term trends and seasonal patterns, the DOW, influenza epidemics and holidays are typically included into the model as they have been identified as potential confounders. In Model 5.1, natural cubic splines were selected to smooth functions of confounding factors that potentially have non-linear relationships with the outcome variable. The choice of lags for the air pollutants and temperatures (lag 0–2 for $PM_{2.5}$ and O_3, lag 0 and lag 1–3 for average dry-bulb temperature and for average dew point temperature) and the degrees of freedom used in this model were selected *a priori* with reference to the literature (Roberts 2004; Ren and Tong 2006; Pattenden et al. 2010; Li et al. 2011). Among the four common averaging times—max 1-h, max 4-h, max 8-h and average 24-h—typically used to monitor ambient O_3 concentrations, max 8-h was selected since it has been recommended by World Health Organization (2000) for detecting O_3-related health problems. The mean of maximum and minimum readings of dry-bulb temperature was selected based on the evidence from a study that found that this parameter was the best, compared to maximum or minimum temperature variables in detecting excess mortality associated with heat exposure in Melbourne (Nicholls et al. 2008).

Identifying influenza epidemics for estimating mortality effects of air pollution in this chapter adopted the method used to control for influenza epidemics in the APHEA-2 project (Touloumi et al. 2004). The method detects influenza epidemics based on daily respiratory death counts. In this analysis, a day in which the 7-day moving average of the respiratory mortality counts exceeded the 90^{th} percentile for the period 1999 to 2007 was set to be an influenza epidemic day. For detecting the effects of air pollution on ED visits, influenza epidemic periods were defined as those for which influenza counts (J09–J11 of ICD-10) exceeded the 90^{th} percentile during the period 1999 to 2008 (Touloumi et al. 2005).

The analyses in this section, as well as in the other sections of this chapter, were made for different disease categories, age groups and seasons. The mortality outcomes were analysed as three disease categories, including all-cause, cardiovascular and respiratory. The ED visits were divided into two categories: cardiovascular and respiratory. Each health outcome was separately analysed according to age group, as shown in Table 5.1. Seasonal analyses were performed to detect health effects attributable to $PM_{2.5}$ for all seasons, the summer (December to February) and the winter (June to August). Analyses for health effects attributable to O_3 were performed for all seasons and for summer. The mortality data for December 2007 were excluded from the analyses due to the reason

explained in Chapter 3. The degrees of freedom used for the seasonal analyses were the same as the all-season analyses.

Table 5.1 Age classification for each health outcome of interest for estimating the health effects of exposure to $PM_{2.5}$ and O_3

Health outcome	Age classification
Mortality	
All-cause	All-ages
	<65 yrs
	65–74 yrs
	≥75 yrs
Cardiovascular	All-ages
	<65 yrs
	65–74 yrs
	≥75 yrs
Respiratory	All-ages
	<75 yrs
	≥75 yrs
ED visits	
Cardiovascular	All-ages
	<65 yrs
	≥65 yrs
Respiratory	All-ages
	0–14 yrs
	15–64 yrs
	≥65 yrs

To examine the potential confounding effects of $PM_{2.5}$ and O_3 in estimating their health risks, the pollutants were also included simultaneously as a co-pollutant in the model. The model that contains both pollutants is referred to as a two-pollutant model, whereas the others outlined above that take account of $PM_{2.5}$ or O_3 separately are referred to as single-pollutant models.

Model 5.1 was fitted using Stata statistical software version 12. The Spline package in R statistical software version 2.15.1 was used to calculate the basis functions of the natural cubic splines. Then the basis functions were imported into Stata and applied in the model to control for time trends.

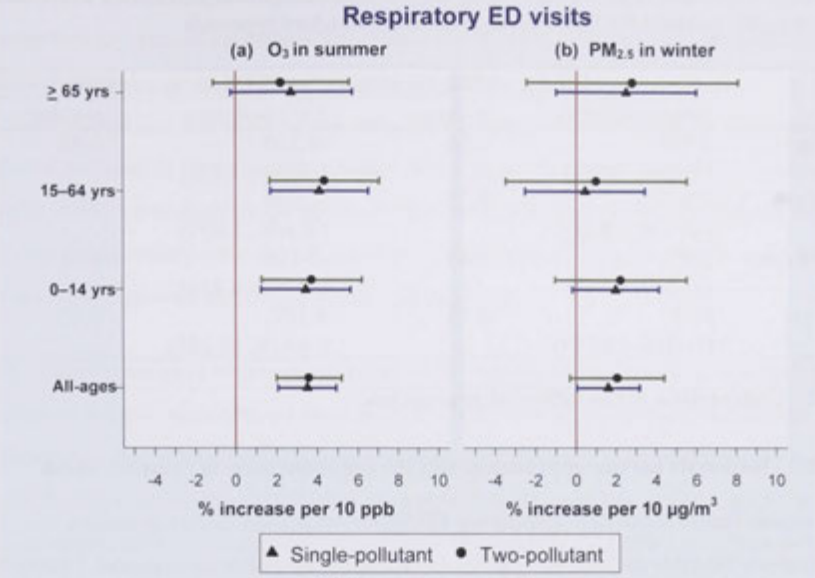
5.2.1.2 Results

Using the standard approach, 'respiratory ED visits' was the only health outcome that showed significant positive associations with the air pollutants considered. No significant

positive associations between $PM_{2.5}$ or O_3 and the mortality outcomes were detected. Relative risk estimates for all health outcomes are presented as percentage changes per 10 $\mu g/m^3$ and 10 ppb increase of $PM_{2.5}$ and O_3 respectively in Appendices C.1 to C.4.

Based on all-seasons analyses, there were no significant positive associations between respiratory ED visits for either pollutant. By season, however, respiratory ED visits was significantly associated with O_3 in the summer (see Figure 5.2a). Such significant associations were apparent in three age classifications—all-ages, 0–14 and 15–64 years—irrespective of whether the results were from the single-pollutant or two-pollutant models. For $PM_{2.5}$, significant positive associations for respiratory ED visits were found only in the winter in the all-ages group, and only when analysed using the single-pollutant model (see Figure 5.2b). Although the associations between $PM_{2.5}$ and respiratory ED visits in the summer were not statistically significant in any age group, the risk estimate found in the all-ages group derived from the single-pollutant model was borderline statistically significant at p=0.06 (see Table 5.2).

Unexpectedly, significant negative associations were found in two health outcomes: all-cause mortality associated with O_3 derived from the two-pollutant model in the 65–74 age group in the summer (-7.38% increase per 10 ppb; 95% CI: -13.73, -0.98) and respiratory ED visits associated with $PM_{2.5}$ derived from the single-pollutant model in the all-season analysis in the 15–64 age group (-1.70% increase per 10 $\mu g/m^3$; 95% CI: -3.39, -0.01). In interpreting results of epidemiological studies of air pollution, negative associations between exposure to air pollution and mortality or morbidity outcomes are mostly speculated to be as a result of model misspecification or inadequate control of confounding variables, rather than the true associations (Katsouyanni et al. 2009; Roberts 2004; Lee et al. 2000). Another possibility that could be used to explain the negative associations is competing risks. This type of situation could occur when frail people are exposed to a severe risk such as a heatwave, thus there are less at−risk people to be hospitalised. The speculation given here for the negative associations is based on toxicological evidence of adverse effects of air pollution on health, even at low levels (Stanek et al. 2011; Alexis et al. 2010). Given the less likely causality of a protective health effect of air pollution, these two health outcomes were not considered further.



Note: (a) per 10 ppb increase for O_3 in the summer and (b) per 10 $\mu g/m^3$ increase for $PM_{2.5}$ in the winter

Figure 5.2 Estimates of percentage changes (95% CI) in respiratory ED visits associated with exposure to $PM_{2.5}$ and O_3 derived from the standard approach

Based on the results described above, respiratory ED visits was the chosen health outcome used further for the purpose of selecting the best approach in the second step. As there were no significant positive associations detected from the first step based on the all-seasons analyses, only seasonal analyses were performed in the second step. For the seasonal analyses, all-age classifications and analyses using both single- and two-pollutant models were performed as follows: i) respiratory ED visits for $PM_{2.5}$ in the summer, ii) respiratory ED visits for $PM_{2.5}$ in the winter and iii) respiratory ED visits for O_3 in the summer.

Table 5.2 Estimates of percentage changes (95% CI) in respiratory ED visits in the summer per 10 μg/m^3 increase for $PM_{2.5}$ derived from the standard approach

	Effects of $PM_{2.5}$ on respiratory ED visits in summer			
	Single-pollutant	p-value	Two-pollutant	p-value
All-ages	3.938 (-0.096, 7.988)	0.06	-0.330 (-4.769, 4.128)	0.88
0–14 yrs	3.107 (-3.376, 9.633)	0.35	-1.540 (-8.686, 5.657)	0.67
15–64 yrs	2.95 (-3.769, 9.644)	0.40	-2.134 (-9.470, 5.256)	0.57
≥65 yrs	6.787 (-1.310, 14.949)	0.10	4.259 (-4.656, 13.255)	0.35

5.2.2 Comparison of the different approaches

5.2.2.1 Rationale for the approaches and the criterion used to compare them

The chosen health outcome—respiratory ED visits—was used further to make a comparison between the four approaches to measuring air pollution exposure. Due to the fact that there is spatial heterogeneity of pollution exposure, particularly for a large city, an approach such as the standard approach that disregards this concern can introduce exposure measurement error. The intention of comparing the standard approach with the nearest and blending approaches was that these two alternative approaches take into consideration the spatial heterogeneity to minimise the exposure measurement error. It can be expected that handling exposure measurement error through these approaches can help improve estimations of the health risks. To address spatial heterogeneity, the unit of analysis has to be shifted from a large geographical level to a smaller one. To apply the SLA, nearest and blending approaches, the selected outcome variable—daily count of respiratory ED visits—and the explanatory variables—air pollution and weather parameters—that were aggregated at the city level in the standard approach were disaggregated to provide the data for each SLA. Apart from the nearest and blending approaches, the SLA approach was designed to be a control in examining the effect of shifting the unit of analysis to address spatial variability in the health data, but not in the air pollution data.

As introduced earlier, the size of the relative risks was selected to evaluate which approach performs best in terms of estimating the relative health risks. The rationale for selecting this criterion was based on the principle of measurement error. In regression analyses, it is well established that when explanatory variables are measured with error, coefficients are attenuated and tend to approach zero (Fuller 1987). Here, therefore, it was hypothesised that relative risks estimated by the approach that can estimate air pollution exposure with the lowest measurement error should move away from the null. In other words, such an approach should provide the largest health risk estimates.

Testing a null hypothesis to determine a statistically significant difference in the magnitude of estimated relative risks derived from the four approaches was not undertaken in the analyses here. This is because the four sets of relative risks were estimated from the same data set. In other words, the four modelling approaches were used to estimate the same effect in reality. In this circumstance, it would be meaningless to apply a null hypothesis test to a parameter derived from the identical data set. This is unlike the typical circumstances that compare independent estimates derived from different data sets where testing for a statistically significant difference is needed.

5.2.2.2 Statistical methods

To model the three approaches as stated above, the spatial unit of analysis was the SLA. For the SLA approach, daily air pollution and weather data were assumed to have no spatial variation (i.e., identical to those used for the standard approach). For the nearest approach, as explained in Chapter 3, the daily exposure to air pollution and weather for a given SLA was estimated by using a measurement from the nearest monitoring station. For the blending approach, the daily exposure to air pollution and weather estimated by the air quality modelling and the blending method described in Chapter 4 in grid cells located inside a given SLA were averaged to represent the daily estimate for that corresponding SLA. A Poisson regression model was fitted for each of the three approaches as shown in Equation 5.2.

$$log\left(\mu_{i,t}\right) =$$
$$\alpha + \beta_1\, pollutant_{i,t,0-2} + s_1\left(avgtemp_{i,t}, 6df\right) + s_2\left(avgtemp_{i,t,1-3}, 6df\right) +$$
$$s_3\left(avgdew_{i,t}, 4df\right) + s_4\left(avgdew_{i,t,1-3}, 4df\right) + s_5(time, 7df\ per\ year) +$$
$$+\boldsymbol{\gamma_1 DOW} + \boldsymbol{\gamma_2 influenza} + \boldsymbol{\gamma_3 holiday} + \sum_{i=1}^{N} \boldsymbol{\gamma_{4,i}}\, \boldsymbol{I(sla = i)} +$$
$$\sum_{i=1}^{N} s_6\,(time, 2df) I(sla = i) \qquad (5.2)$$

Building on Model 5.1, two terms accounting for spatial variations in air pollutants and temperatures as well as health outcomes were added to construct Model 5.2. The first term is a vector of coefficient (γ_4) to control differences among the SLAs (sla), such as the size of populations, population characteristics and socio-economic conditions. The other term is an interaction between the indicator variable for SLA (sla) and a smoothing function of calendar time (s_6) to control for differences in outcome rates among the SLAs that change over time. The parameters of the health outcome, air pollution, dry-bulb temperature and dew point temperature have the subscript i to indicate specific variation of these variables for each SLA. Model 5.2 was fitted with varying degrees of freedom from one to seven for the time variable of that interaction term in order to choose how many degrees of freedom (df) provided the best fitted model. Model fit was justified on the basis of minimising the Akaike's information criterion (AIC)—an indicator of the goodness of fit of a statistical model. Two degrees of freedom in s_6 was identified as providing the best fit for Model 5.2.

5.2.2.3 Results

Figures 5.3 and 5.4 compare relative risk estimates for respiratory ED visits associated with $PM_{2.5}$ and O_3 respectively derived from the four approaches. Based on the criterion, it can be seen that the largest estimates were mostly found, particularly in the summer, when the blending approach was applied to estimate exposures to air pollution and weather. In most cases, the nearest approach appeared to provide the smallest estimates. The size of risk estimates derived from the SLA approach was identical to those from the standard approach. Among the different age groups, the degree of the difference in risk estimates derived from the blending approach compared to the other approaches was most noticeable in the elderly in summer for both pollutants, particularly in the single-pollutant model. For winter, adding the dimension of spatial variation with the use of the nearest and blending approaches in Model 5.2 did not appreciably change the estimate of the association between $PM_{2.5}$ levels and adverse health outcomes (see Figure 5.3, right panel). All

estimates in Figures 5.3 and 5.4 are also presented in tabular form in Appendices C.5 and C.6.

There was little difference in the association between O_3 in summer and ED visits for respiratory outcomes for the two-pollutant model, compared with the single-pollutant model, regardless of the modelling approach used. In contrast, estimates for $PM_{2.5}$ in summer derived by having O_3 as a co-pollutant in the model were weaker than modelling this pollutant alone. The same pattern was found across the four age classifications. In the winter, risk estimates associated with $PM_{2.5}$ derived from the single and two-pollutant models were only slightly different.

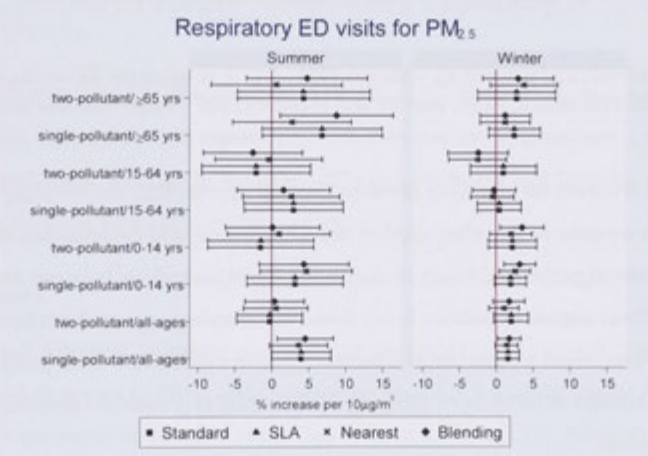


Figure 5.3 Estimates of percentage changes (95% CI) in respiratory ED visits derived from four approaches (standard, SLA, nearest and blending) per 10 μg/m3 increase for $PM_{2.5}$

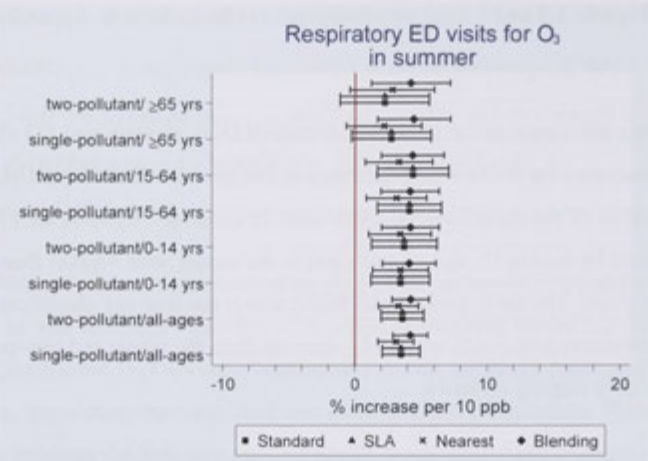


Figure 5.4 Estimates of percentage changes (95% CI) in respiratory ED visits derived from four approaches (standard, SLA, nearest and blending) per 10 ppb increase in O_3 in the summer

Based on the criterion for choosing between the four approaches, the blending approach appears to be superior to the other approaches. Discussions and explanations of why the blending approach performed better in estimating the relationships between air pollution and health effects appear in Section 5.2.4. Since the blending approach performed best, it was used to re-estimate all associations between health outcomes and air pollution. The health risk estimates derived from applying the blending approach are presented in the following section.

5.2.3 Estimates of the relationships between acute health outcomes and air pollution

5.2.3.1 Statistical methods

First, Model 5.2 with the use of the blending approach was applied to obtain risk estimates associated with air pollution for all health outcomes. Estimates of percentage change based on fitting Model 5.2 for all health endpoints are presented in Appendices C.7 to C.10. Due to having many (over 300) parameters in the model, the model failed to converge when it was fitted for the health outcomes with few events, particularly seasonal analyses for respiratory mortality (see Appendices C.7 and C.8). Since the largest number of parameters was from the interaction term between *sla* and *time* (78 SLA*2df for time=156

parameters[2]), a reduction of the df for time from 2df to 1df was tested. This change in the number of df removed 78 parameters from the model and as a result the model was successfully fitted for all health outcomes. Thus, for the purpose of obtaining a complete set of risk estimates for all health outcomes, 2df for the *time* variable interacting with the *sla* variable in Model 5.2 was lowered to 1df and used from this point onward. Below is the notation of the 1df model, referred to as the 'basic model', which was used further in the following chapter in estimating the relative risks for the health effects of air pollution modified by temperature.

$$\log(\mu_{i,t}) = \alpha + \beta_1\, pollutant_{i,t,0-2} + \sum_{i=1}^{N} \beta_2\,(time) I(sla = i) + s_1(avgtemp_{i,t}, 6df) + s_2(avgtemp_{i,t,1-3}, 6df) + s_3(avgdew_{i,t}, 4df) + s_4(avgdew_{i,t,1-3}, 4df) + s_5(time, 7df\ per\ year) + \boldsymbol{\gamma_1 DOW} + \boldsymbol{\gamma_2 influenza} + \boldsymbol{\gamma_3 holiday} + \sum_{i=1}^{N} \boldsymbol{\gamma_{4,i}}\, \boldsymbol{I(sla = i)}, \quad (5.3)$$

Due to the reduction of df as described, s_6 in Equation 5.2 becomes β_2 in Equation 5.3 as it becomes linear. All notations in Equation 5.3 are the same as in Equation 5.2, except β_2, which denotes a linear function of the effect of long-term trends and seasonal cycles in the form of calendar time for each indicator variable *sla*.

5.2.3.2 Results

Figures 5.5 to 5.8 present estimates of percentage change in daily mortality and ED visits associated with exposure to $PM_{2.5}$ and O_3 based on Model 5.3 with the use of the blending approach (see estimates in tabular form in Appendices C.11 to C.14). When the blending approach was used to re-estimate the associations between all health outcomes and the air pollutants considered, most of the mortality outcomes, stratified by disease categories, age groups and seasons, did not have a statistically significant association with exposure to $PM_{2.5}$ and O_3. Nevertheless an association between all-cause mortality in the age group 65–74 years and exposure to $PM_{2.5}$ in the winter in the single-pollutant model was statistically significant (see Figure 5.5a). Likewise an association between cardiovascular mortality in the age group 65–74 years and exposure to $PM_{2.5}$ in the winter in both the single- and two-pollutant models was borderline statistically significant (see Figure 5.5b). For the ED visits outcomes, apart from the health outcomes significantly associated with

[2] There are 79 SLAs in the Melbourne Region. However, one SLA, 205607456, in the interaction term between *time* and *sla* was omitted when Model 5.2 was fitted due to insufficient events for the outcome variable for that SLA. Therefore, there were only 78 parameters for the *sla* variable.

the air pollutants previously presented, applying the blending approach resulted in detection of significant associations of some further respiratory and cardiovascular ED visits outcomes. Examples of these significant outcomes include the association between cardiovascular ED visits outcome and $PM_{2.5}$ in all-ages group in the two-pollutant model in the winter (see Figure 5.7a) and the associations between respiratory ED visits and O_3 in all-ages, 0–14 years and 15–64 years in the single-pollutant model in all seasons (see Figure 5.8 b).

Considering the risk estimates derived from the single- and two-pollutant models, no difference was apparent for O_3, irrespective of the seasons analysed. The risk estimates for $PM_{2.5}$ appeared not to be different when the data from all seasons were included in the analysis, regardless of the health outcomes considered. Nonetheless including O_3 into the $PM_{2.5}$ two-pollutant model had an impact on the risk estimations when the data were analysed by season. In addition such impact appeared to be stronger when the risk estimates were statistically significant. For example, there was no distinguishable difference between the risk estimates associated with $PM_{2.5}$ for respiratory mortality derived from the single- and two-pollutants models in the summer (see Figure 5.5c). However, the association between $PM_{2.5}$ and respiratory ED visits derived from the two-pollutant model in summer became statistically non-significant and was of lower magnitude compared to the effect derived from the single-pollutant model (see Figure 5.7 b). An overall observation from the results in this section is that adding the O_3 variable into the $PM_{2.5}$ two-pollutant model attenuated the risk estimates associated with $PM_{2.5}$ in the summer, but increased the risk estimates for $PM_{2.5}$ in the winter.

In terms of the difference in risk estimates among the age groups, the association between $PM_{2.5}$ and ED visits were generally greater and statistically significant in the elderly and children. There was a stronger, and borderline statistically significant, association between $PM_{2.5}$ and cardiovascular ED visits derived from the two-pollutant model in the winter for the >65 years age group compared to those <65 years (see Figure 5.7a). For the association between $PM_{2.5}$ and respiratory RD visits in the summer, stronger estimates were apparent in the elderly, while the estimates for the effect of $PM_{2.5}$ in the winter in children were relatively similar to those for the elderly, and higher than those for the middle aged group (see Figure 5.7b). Such a difference between the age groups was not apparent for the association between O_3 and respiratory ED visits (see Figure 5.8b).

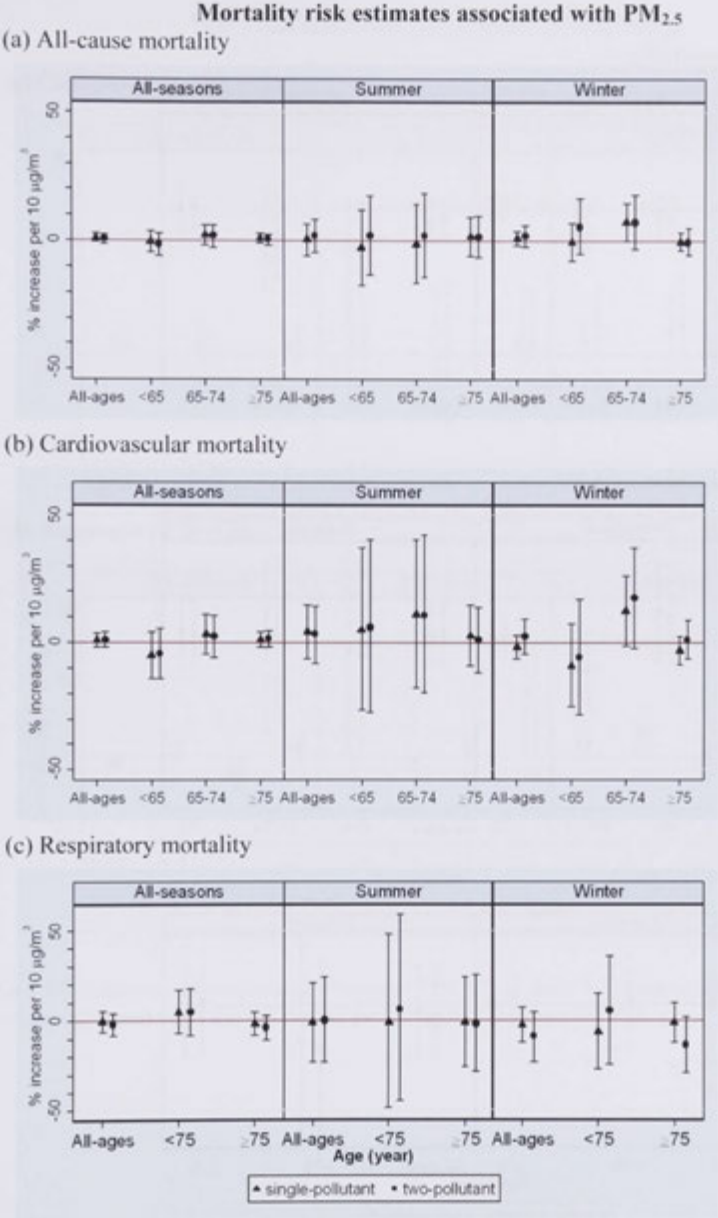
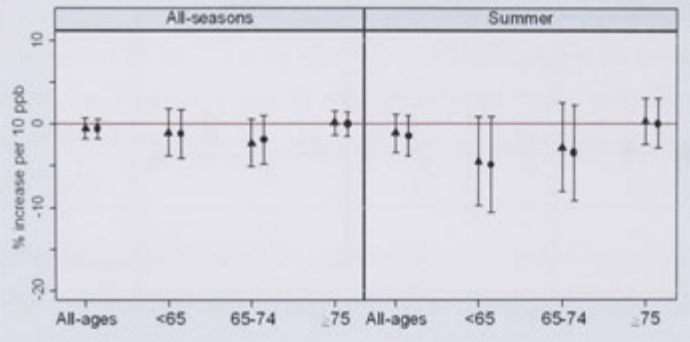


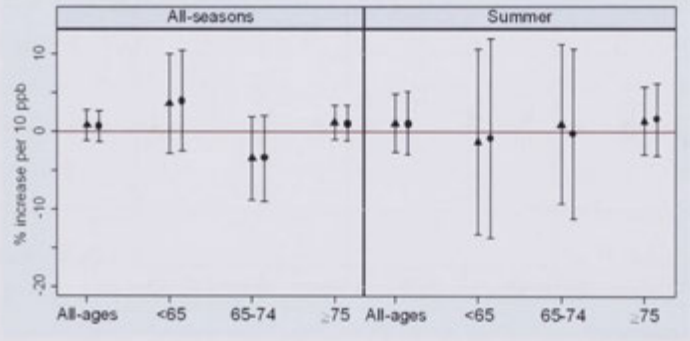
Figure 5.5 Estimates of percentage changes (95% CI) in daily mortality derived from the blending approach with the 1df model per 10 $\mu g/m^3$ increase for $PM_{2.5}$

Mortality risk estimates associated with O_3

(a) All-cause mortality



(b) Cardiovascular mortality



(c) Respiratory mortality

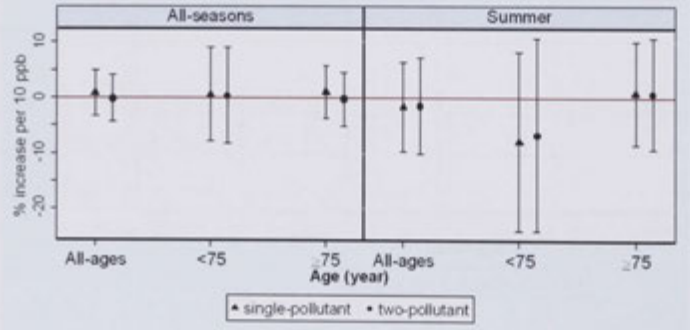
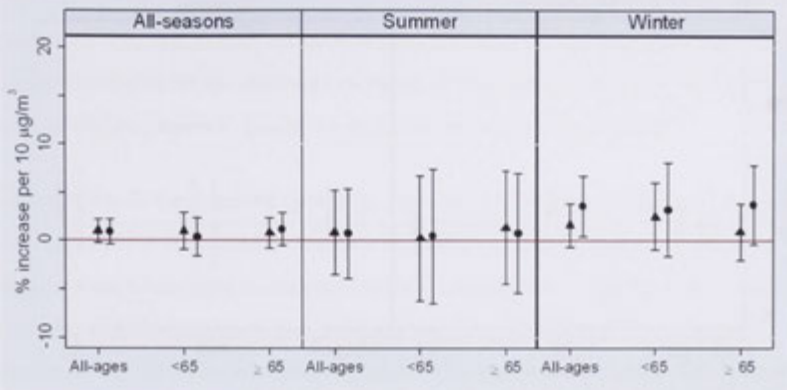


Figure 5.6 Estimates of percentage changes (95% CI) in daily mortality derived from the blending approach with the 1df model per 10 ppb increase for O_3

ED visits risk estimates associated with $PM_{2.5}$

(a) Cardiovascular ED visits



(b) Respiratory ED visits

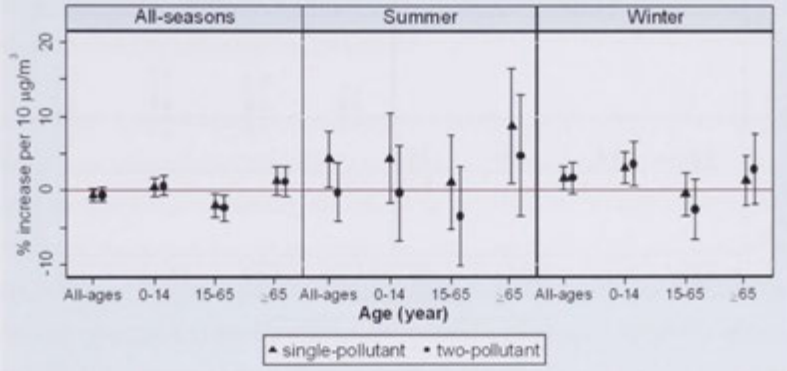
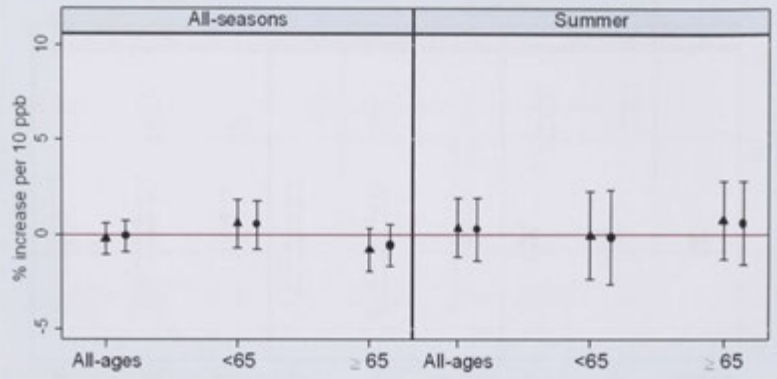


Figure 5.7 Estimates of percentage changes (95% CI) in daily ED visits derived from the blending approach with the 1df model per 10 $\mu g/m^3$ increase for $PM_{2.5}$

ED visits risk estimates associated with O_3

(a) Cardiovascular ED visits



(b) Respiratory ED visits

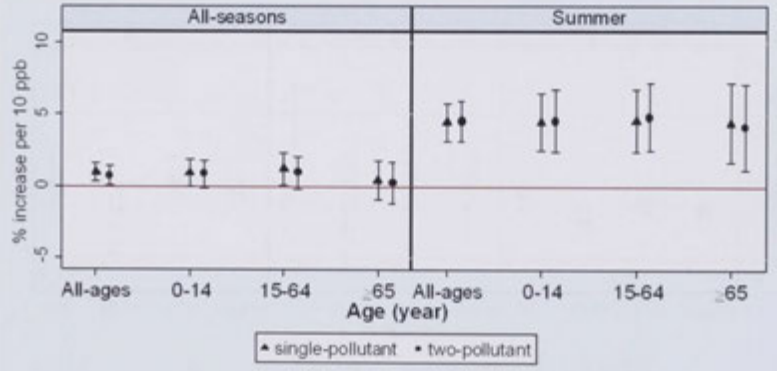


Figure 5.8 Estimates of percentage changes (95% CI) in daily ED visits derived from the blending approach with the 1df model per 10 ppb increase for O_3

5.2.4 Discussion

Among the four approaches examined for estimating health risks associated with exposure to $PM_{2.5}$ and O_3 in the Melbourne Region, the blending approach performed better than the others based on the criterion of the size of risk estimates. Overall the blending approach gave larger estimates for the associations between respiratory ED visits—the health outcome of choice chosen to gauge the performance—and both pollutants.

With application of the standard approach, there was no significant positive association between exposure to $PM_{2.5}$ and O_3 and any mortality outcomes. However, when the blending approach was used to re-estimate the mortality risks, significant positive associations, including borderline significant, were found between all-cause and cardiovascular mortality in the elderly aged 65–74 years and exposure to $PM_{2.5}$ in the winter. Likewise, compared to the standard approach, a greater number of significant positive associations were detected between ED visits outcomes and exposure to $PM_{2.5}$ and O_3 when the blending approach was employed.

In this analysis, a seasonal effect was apparent, whereby associations with adverse health outcomes were generally stronger for O_3 and $PM_{2.5}$ in the summer and $PM_{2.5}$ in the winter. Age was another factor influencing the size of the associations. Mostly children and the elderly in the Melbourne Region seemed to be more vulnerable to air pollution exposure than the other age groups as discerned by the greater size of the risk estimates, compared to the other age groups. To identify whether there is any statistically significant difference between the risk estimates across the age groups, a number of tests will be undertaken and described in Chapter 6.

5.2.4.1 Estimates of the adverse health effects associated with air pollution in the Melbourne Region

5.2.4.1.1 Estimates of the mortality effects

The analyses presented here demonstrated that very few mortality health outcomes had a positive and statistically significant association with $PM_{2.5}$ exposure. Further, none of the mortality outcomes investigated had a positive significant association with O_3. This is in contrast to the results of previous studies in Australia and overseas. Three studies have estimated relationships between exposure to air pollution and mortality and included results for Melbourne (Simpson et al. 2005a; Environmental Protection and Heritage

Council 2010; Simpson et al. 2000). Of these three studies, the SPIRT and EPHC studies did not particularly focus on health effects and air pollution in Melbourne (Simpson et al. 2005a; Environmental Protection and Heritage Council 2010). Rather, their ultimate objective was to incorporate the results for Melbourne into an estimation of mortality effects of air pollution for Australia through applying the multi-city approach, as described in Chapter 2. The third study did have a particular focus on Melbourne (Simpson et al. 2000), using a single-site approach. Among these three studies, significant associations between mortality outcomes and exposure to air pollution in Melbourne were found in the EPHC study and the Melbourne-focused study by Simpson et al. (2000). In the SPIRT study, all-cause mortality was not significantly associated with exposure to air pollution in Melbourne, while individual results specific to each city including Melbourne were not reported for the other two mortality outcomes.

The divergent findings in relation to the association between mortality and exposure to $PM_{2.5}$ and O_3 may be explained by whether extreme air pollution episodes were included in the analyses or not. The EPHC study and that reported by Simpson et al. (2000) retained these data and found a significant association between air pollution and mortality, possibly reflecting the health effects of extreme air pollution events. The analyses for both this thesis and for the SPIRT study removed days in which PM or nephelometer concentrations were high and neither study found a significant association between $PM_{2.5}$ or O_3 and all-cause mortality. This issue is examined in detail in Section 5.3 through reanalysing the data with the inclusion of days with evidence of bushfires and dust storms.

Another possible explanation for the non-detection of the mortality effects reported here relates to limiting the analysis to one city, with the result of insufficient statistical power. This issue is critical for studying the mortality effect of air pollution where the effect sizes may be relatively small. Even in a moderately large city such as Melbourne (population 4.1 million in 2011), the average daily all-cause mortality count over the study period was only around 50. As reviewed in Chapter 2, the challenge of insufficient sample size to detect a small effect when confining analyses to a single city has driven epidemiological studies of air pollution over the past decade to adopt the multi-city approach.

An additional explanation is exposure measurement error. With the application of the blending approach, it was believed, as shown in this chapter based on the increased relative health risks, that measurement error was reduced. However, even with the reduction of

measurement error by using the blending approach, it may be possible that to a certain extent measurement error still exists. Unless using data derived from measuring individual exposures, it is still possible that detecting no mortality effect is due to measurement error.

5.2.4.1.2 Estimates of the morbidity effects

Previous studies have found significant associations between exposure to air pollution and respiratory morbidity in the major Australian capital cities, as reviewed in Chapter 2. The positive associations between respiratory ED visits with exposure to $PM_{2.5}$ and O_3 found here in children in the summer in the single-pollutant model, which are stronger than those found in the winter, are similar to the results of a study in Sydney (Jalaludin et al. 2008). That study reported a 0.9% and a 1.5% increase per interquartile range (IQR) for $PM_{2.5}$ 24-h (4.4 $\mu g/m^3$) and O_3 max 1-h (20.5 ppb) respectively at lag 0 in ED visits for asthma in the 1 to 14 year age group in warm months, with a stronger effect for the warm versus the cool months. This is in contrast to a study in Perth that did not find significant increases in asthma ED visits associated with PM_{10} and O_3 exposure in children aged 0–19 years, although such increases were found for CO and NO_2 (Pereira et al. 2010).

Toxicological studies confirm that exposure to PM has adverse effects on the cardiovascular system by targeting the autonomic nervous system and inducing systemic oxidative stress and vascular inflammation (Martinelli et al. 2013; Stanek et al. 2011). Such a malfunction of the cardiovascular system can be played out into different forms of cardiovascular diseases such as congestive heart failure, IHD and stroke, as previously shown in epidemiological studies (Ruckerl et al. 2011). Similar to some epidemiological studies conducted in Melbourne, the analysis here provides evidence of stronger and significant cardiovascular effects in the winter compared to the summer. For example, the EPA Victoria (2001) reported a 4.9% increase in emergency hospital admissions for cardiovascular disease in all-ages at a 1-day lag per $1x10^{-4}$ m^{-1} increase in Bsp 24-h (equivalent to 15$\mu g/m^3$ $PM_{2.5}$) in cool seasons, which was stronger than a 3.0% increase in warm seasons. Similarly, the EPHC study also reported significant increases in the risk of emergency hospital admissions for some cardiovascular diseases in Melbourne in association with exposure to $PM_{2.5}$ and Bsp only in cool seasons (Environmental Protection and Heritage Council 2010). The null results for exposure to $PM_{2.5}$ and cardiovascular ED visits in the summer presented in this chapter may be related to the exclusion of air pollution readings on days with bushfires and dust storms. The re-analysis conducted in

Section 5.3 provides some insight into an underlying cause of the null results for the summer found here.

5.2.4.2 Single vs. two-pollutant models

The correlations between air pollutants and to what extent they impact on the interpretation of the relationship between multiple ambient air pollutants and health effects have been of concern in epidemiological studies of air pollution. As a result of the correlations, some of the issues including confounding, surrogates or proxy indicators, multicollinearity and interactions among pollutants have been widely debated and researched (Kim et al. 2007; Bateson et al. 2007). From this literature, this section applies insight acquired from past studies on two issues—confounding and surrogates—that can be used in discussing the results obtained from the single- and two-pollutant models of O_3 and $PM_{2.5}$ reported in the previous sections.

Many studies have considered gaseous and particle pollutants as confounders as most of these pollutants have causal effects on health and their ambient concentrations are highly correlated due to their emission from the same sources. In estimating the relationships between air pollution and health effects by time-series studies, a common approach to deal with the issue of confounding is to include co-pollutants into the same model with the air pollutant of interest for adjustment. Studies taking such an approach have shown that risk estimates for effects of O_3 or PM on health outcomes derived from two-pollutant models were often robust to adjustment of the other co-pollutant (Bell et al. 2004; Samet J. M et al. 2000). For studies using a case crossover method, controlling for other pollutants is done by matching on concentrations of the potential co-pollutant(s) between case days and control days. With further regard to this issue, Schwartz (2004) investigated the role of CO, O_3 and SO_2 as confounders of an association between PM_{10} and mortality using the NMMAPS database. In that analysis, the co-pollutants, including O_3, did not confound the observed PM_{10} association. Based on the same database, results from a study by Bell et al. (2007b) suggested that $PM_{2.5}$ was not a potential confounder of an association between O_3 and mortality. However, a study by Franklin and Schwartz (2008) found that the sulphate component of $PM_{2.5}$, which is formed through similar photochemical processes as for O_3, could be a potential confounder for a relationship between O_3 and mortality. This was suggested by a dramatic decrease of the apparent relationship in the model that adjusted for the sulphate component.

For the analyses conducted here to compare the risk estimates derived from the single- and two-pollutant models, the knowledge of the confounding effects between O_3 and $PM_{2.5}$ from the studies stated above might help explain the results. As presented in Chapter 3, the ambient concentrations of O_3 and $PM_{2.5}$ in the Melbourne Region have a moderate positive correlation in the summer (correlation coefficients ~ 0.4 to 0.5) and a moderate negative correlation in the winter (correlation coefficients ~ -0.5 to -0.6). Because of the correlations, it is likely that these two pollutants are potential confounders of the health effects of each other. The attenuation of the statistically significant increased risks of respiratory ED visits associated with $PM_{2.5}$ found in the two-pollutant models in the summer (see Figure 5.7b), may indicate evidence of this. Similarly, the increase in size and significance of the risks of cardiovascular mortality and ED visits associated with $PM_{2.5}$ in the winter (see Figures 5.5b and 5.7a) may be related to the negative correlation between $PM_{2.5}$ and O_3.

In contrast to the results above, the inclusion of $PM_{2.5}$ into the two-pollutant model of O_3 did not alter the magnitude or statistical significance of the increased risks for respiratory ED visits related to O_3 in the summer, despite the positive correlation between O_3 and $PM_{2.5}$ concentrations. The unchanged risks associated with O_3 with the adjustment for $PM_{2.5}$ may be explained by reference to the results observed by Franklin and Schwartz (2008). In that study, an association between mortality and ambient O_3 was robust to the control of total $PM_{2.5}$ mass, but not for the sulphate component of $PM_{2.5}$ as stated above.

Apart from the confounding effects, ambient gaseous pollutants have been hypothesised to be surrogates of PM pollutants in terms of their effects on health. Sarnat et al. (2001) hypothesised that for gaseous pollutants to be confounders of the health effects of $PM_{2.5}$, not only did their ambient concentrations need to be correlated, but there also needed to be a correlation between the personal exposures. That study demonstrated that the concentrations of ambient gaseous pollutants in Baltimore, Maryland, had a low correlation with their corresponding personal exposures, but had a moderate correlation with personal exposures to $PM_{2.5}$. The authors, therefore, concluded that adverse health effects associated with ambient concentrations of some gaseous pollutants such as CO and NO_2, in which causal relationships seemed less likely, might in fact be an indication of a health effect of personal exposure to $PM_{2.5}$. In other words, the findings from that study suggested that gaseous pollutants could act as surrogates for $PM_{2.5}$ pollutants. One of the underlying reasons to explain the relationships between personal and ambient

concentrations of gaseous and PM pollutants may be associated with measurement error. This issue has also been increasingly investigated by many studies, as is discussed in the following section.

The concept of ambient O_3 concentration being a surrogate of personal exposure to $PM_{2.5}$ can help explain why the $PM_{2.5}$ association was attenuated in the two-pollutant model (i.e., controlling for O_3), particularly in the summer. It might be possible that $PM_{2.5}$ and O_3 are not confounders of each other, but rather that O_3 serves as a surrogate for the association between $PM_{2.5}$ and the respiratory outcomes. Generally, ambient O_3 concentrations have more spatial homogeneity than $PM_{2.5}$ as O_3 is a secondary pollutant. This characteristic of O_3 makes ambient concentrations more representative of the true population exposure, less subject to measurement errors and thus a better predictor of the adverse health effects, compared to $PM_{2.5}$. Moreover a network monitoring ambient O_3 in a city has generally a better coverage than a network monitoring ambient $PM_{2.5}$. This again makes O_3 less sensitive to measurement error. To test the proposition that O_3 is acting as a surrogate for personal exposure to $PM_{2.5}$ in terms of health effects in the Melbourne Region requires information on correlations between ambient concentrations and personal exposures of these two pollutants. Unfortunately, such information is still limited in Australia but would be worthy of future investigation.

5.2.4.3 The differences between the four approaches

The consistently stronger associations between exposure to $PM_{2.5}$ and O_3 and respiratory ED visits estimated by the blending approach may be explained by the interplay of two factors: i) the inclusion of variations at the within-city scale through changing the unit of analysis from city to SLA and ii) the ability of the blending method to provide better estimates of air pollution exposures at finer resolution.

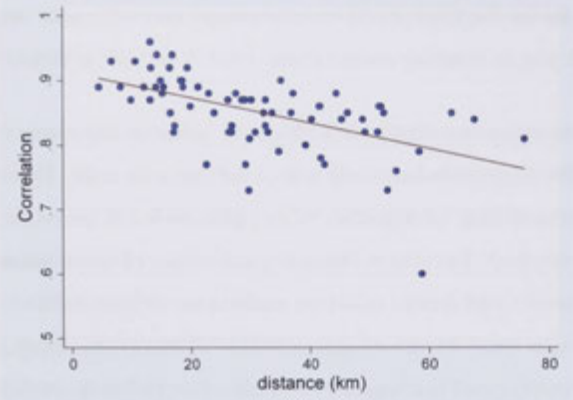
In relation to the first factor, Bateson et al. (2007) remarked that changing to a smaller unit of aggregation for epidemiological analyses to match with the scale that observed health endpoints were generated had potential to improve estimates of the health effects of air pollution. Results of two studies in the US, which observed increased sizes of relative health risks of air pollution when using a small unit of analysis (at the level of the county), were used as evidence to support such a proposal (Willis et al. 2003; Jerrett and Finkelstein 2005). As can be seen from the identical risk estimates derived from applying the standard and SLA approaches (see Figures 5.3 and 5.4), the change in spatial level from city to SLA

alone had a little impact on the effect estimates for respiratory ED visits. This suggests that in these analyses, the second factor, the finer resolution of exposure measurements, may have a more important role to play in reducing measurement error than the first factor.

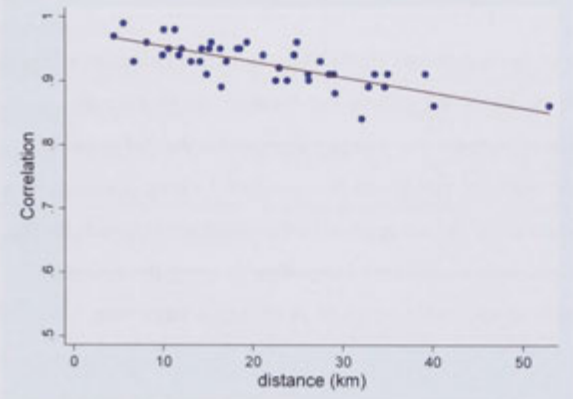
For the second factor, the more accurate estimates of ambient air pollution exposures at the SLA level generated by the blending method can help reduce the exposure error. This type of error potentially stems from assuming homogeneity of air pollution levels across the study area. Such an error is commonly found in epidemiological studies of air pollution that apply conventional approaches and depend solely on measurements from fixed monitors. The Melbourne Region spans an area of approximately 7500 km^2, therefore, it is likely that there is some degree of spatial heterogeneity of ambient air pollution, even for $PM_{2.5}$ and O_3 where the gradient levels across the city are not substantial, compared to other primary pollutants.

Figure 5.9 shows correlations of measurements (both $PM_{2.5}$ and O_3) of all pairs of the fixed monitors in Melbourne versus distances; the greater the distance, the smaller the correlation. The greatest distances between the paired monitors for the $PM_{2.5}$ and O_3 networks are 53 and 76 km respectively, which can be considered a long distance. As a result, using the daily averaged measurements applied in the standard approach for the entire city is unlikely to be as accurate a measure of exposure as using the estimates acquired from the blending method assigned to each SLA in such a large area.

(a) $PM_{2.5}$



(b) O_3



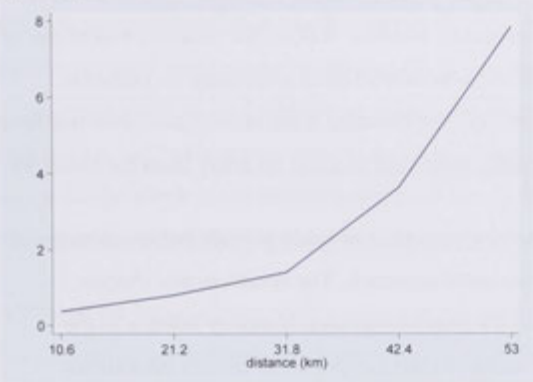
Note: Each dot represents a pair of monitoring stations

Figure 5.9 Correlations between daily ambient concentrations of pairs of monitoring stations and distances in the Melbourne Region for (a) $PM_{2.5}$ and (b) O_3

Figure 5.10 shows semi-variogram plots of aggregated $PM_{2.5}$ and O_3 concentrations over the decadal period measured in the monitoring network in the Melbourne Region. The plots reiterate the information shown in Figure 5.9. As expected, the variance of $PM_{2.5}$ concentrations increases with the greater distance of the monitors. The range of semi-variogram for $PM_{2.5}$ is approximately at 30 km with the sill of approximately 2. In contrast, the variance of O_3 decreases with the greater distance of the monitors. This can be explained by the characteristic of O_3 which is a secondary pollutant. The level of O_3 in the city centre is generally low while the level of the pollutant is higher at the outskirts of the city. At the medium range (the distance within the range of the semi-variogram where the

line is relatively flat), the variance is larger because it reflects the greater difference of low and high concentrations of O_3 measured by the monitors located at different settings (the centre and the periphery of the city). At the great range (the distance beyond the range of the semi-variogram where the steepness of the line is high), the variance becomes smaller as this is the variance between high concentrations of O_3 measured from only monitors located at the periphery of the city.

(a) $PM_{2.5}$



(b) O_3

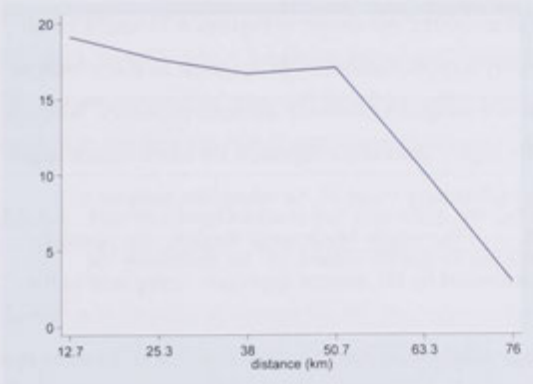


Figure 5.10 Semi-variogram of (a) $PM_{2.5}$ and (b) O_3 concentrations measured from the monitoring network in the Melbourne Region

Again, due to the large span of the Melbourne Region, using the measure that relies on the proximate station as applied in the nearest approach may not be capable of reducing the exposure error to the level that can enhance estimation of relative risks. For example, in estimating $PM_{2.5}$ concentrations through the nearest approach in this chapter, there were 18 SLAs out of 79 in which the distances between their centroids to the nearest stations exceeded 20 km. It is apparent that with the use of the nearest approach, there were not sufficiently accurate estimates of ambient air pollution for some parts of the city. In contrast to the nearest approach, the estimates of air pollution exposures generated from the blending method do not rely on a particular monitor. Rather, the measure makes use of observations of the monitors located in the area of interest in estimating air pollution concentrations for each grid cell. In addition, the blending method also uses estimates from air quality modelling, which is particularly beneficial to areas far away from the monitors.

It is reasonable to expect that using the nearest approach could provide better estimates of relative risks of air pollution than the standard approach. The results in this chapter, however, show that the performance of the nearest approach is mostly inferior to the standard approach. Among methods that have been developed to capture intra-urban differences in air pollution exposures, the proximity technique applied in the nearest approach is the simplest, while the method applied in the blending approach is among one of the most complex methods (Jerrett et al. 2005). As shown in Figures 4.11 and 4.12 of Chapter 4, it is possible that there are only a small number of SLAs, such as those located in inner SLAs, where the nearest approach assigns a relatively accurate exposure. Whereas this approach may introduce potentially highly inaccurate exposure for more distant areas that are the majority of the study area, particularly outer SLAs where the number of monitors is limited. From the perspective of the whole Melbourne Region, this possibly results in a poorer overall true effect estimated by the nearest approach, compared to the standard approach.

The results of the increased magnitude of the relative risks estimated using the blending approach reported here are comparable with a percentage reduction in risk ratios found in a simulation study examining an impact of measurement error on estimations of health risk of air pollution in time-series studies (Goldman et al. 2011). In the study by Goldman et al. (2011), with an error related to spatial variability added to the true exposure, a 2% to 31% reduction in relative risks per unit of measurement was estimated. According to the current analyses, if discarding negative coefficients that are not biologically plausible, the

percentage difference in relative risks for respiratory ED visits associated with O_3 and $PM_{2.5}$ between the standard and blending approaches ranges from 1% to 47% per 1 $\mu g/m^3$ for $PM_{2.5}$ and per 1 ppb for O_3.

The smaller differences between the four approaches for the estimates of percentage changes in respiratory ED visits associated with $PM_{2.5}$ exposure in the winter than in the summer, as shown in Figure 5.3, are possibly suggestive of the variation in exposures to ambient air pollution by season. In winter, when people spend a larger proportion of time indoors, using ambient measurements from fixed monitors or an air quality model may poorly represent true personal exposure. Therefore, applying the blending approach, which is generally useful to deal with the exposure error caused by spatial heterogeneity, may not particularly useful for winter data. In summer, when ambient exposure is closer to personal exposure, the merit of the blending approach in correcting the exposure error stemming from spatial heterogeneity is more pronounced.

Although the blending approach is useful in terms of calculating true estimates of health risks, it has a crucial limitation in the complexity of the methods involved, as discussed in Chapter 4. In practice, for other cities like Melbourne that have a good monitoring network, the standard approach is still an efficient tool, particularly for estimating health risks related to secondary pollutants, including O_3 and secondary components of PM, to primarily identify which health outcomes are significantly associated with the pollutants. Then, a more complex approach that takes into account spatial variability of the city can be applied to confirm the significant associations and estimate their true health risks.

5.2.4.4 Further implications for detecting the interaction effects of temperature and air pollution on the health effects of air pollution

Based on the results of comparing the four approaches, which suggest that the blending was superior in terms of detecting the health risks of air pollution, this approach will be applied further in the following chapter to detect and estimate the interaction effects of temperature and air pollution on health. As a consequence of the superior performance of this approach, it should be capable of providing better estimates of the health risks when the interaction effects of temperature and air pollution are considered in modelling air pollution and health for the time-series studies.

5.3 The inclusion of data on bushfire and dust storm days and its impact on the analyses

As briefly discussed in Section 5.2.4, it was suspected that the restricted detection of positive and significant associations between mortality and air pollution exposure in the Melbourne Region may be related to whether or not the air pollution data on extreme air pollution episodes were included in the analyses. In this section, this proposition was first explored assuming a linear relationship between health effects and air pollution exposure. In the second part of this section, a non-linear relationship of $PM_{2.5}$ to health risk that is likely to result from the difference in $PM_{2.5}$ compositions between normal days and days with extreme air pollution episodes outlined in Chapter 1 was evaluated. This aspect was considered not only to justify the exclusion of data on bushfire and dust storm days applied to the analyses in this chapter, but also to help explain the limited detection of significant mortality association reported earlier.

5.3.1 Linear relationship assumption

Model 5.1 was refitted to re-analyse the data using the standard approach in the single-pollutant model and including the $PM_{2.5}$ and O_3 readings on bushfire and dust storm days. Only the data for all seasons and the summer were re-analysed as the extreme air pollution episodes previously excluded did not occur in the winter. Results are presented only for the all-ages group.

For the mortality associations, results from the re-analysis (see Figure 5.11a) show that the associations between exposure to $PM_{2.5}$ and all-cause and cardiovascular mortality in the all-ages group became stronger and were statistically significant when the readings on bushfire and dust storm days were brought back into the analyses. Also, the risk estimates between respiratory mortality and $PM_{2.5}$ were greater with a narrower CI in this re-analysis. Compared to the associations between $PM_{2.5}$ and mortality, including the data on extreme air pollution episodes appears to have lesser impact on the association between mortality and O_3 (see Figure 5.11b).

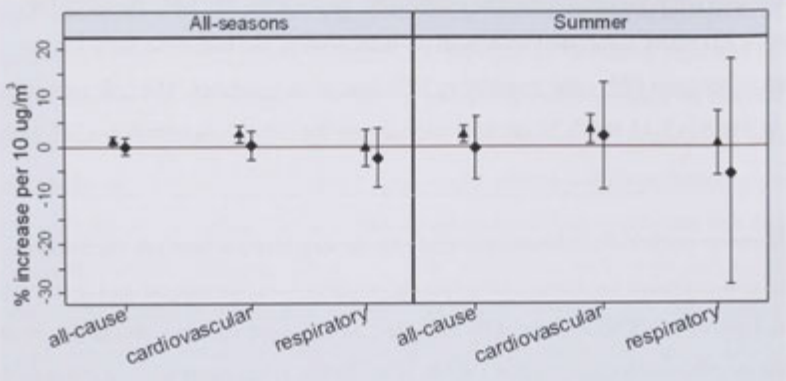
Figure 5.12 shows that including air pollution readings on days with extreme air pollution episodes in the re-analysis generally reduced the CI of the associations between ED visits and exposure to $PM_{2.5}$. The risk estimates for cardiovascular ED visits associated with $PM_{2.5}$ and O_3 based on the re-analysis were mostly of greater magnitude than in the original

analysis. There was one exception in which the size of the risk estimate between $PM_{2.5}$ and cardiovascular ED visits in the summer was weaker with inclusion of the extreme episodes, but it changed from non-significant to significant. In contrast, the risk estimates for respiratory ED visits associated with both pollutants were weaker, especially for the association between $PM_{2.5}$ and respiratory ED visits in the summer. The risk estimates shown in Figures 5.11 and 5.12 are presented in tabular form in Appendices C.15 and C.16.

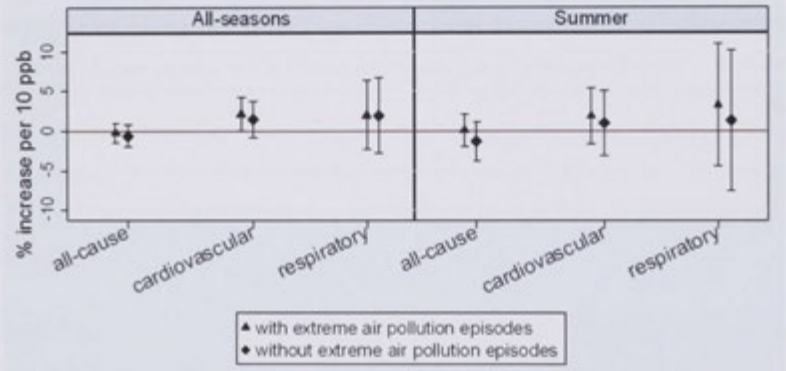
The difference in the risk estimates associated with air pollution between the analyses that included and excluded the data of air pollution on days with extreme air pollution episodes presented above could be explained by the effect of exposure variance on the power of detection in epidemiological studies (White et al. 1994). It has been well established that a large sample size is required to be capable of identifying an association between a disease and an exposure in a population with a small variance of exposure. To increase the power of detection under the condition of a small sample size, in particular when measurement error exists, it is more favourable to select a population that has a large variability of exposure.

Mortality risk estimates associated with $PM_{2.5}$ and O_3

(a) $PM_{2.5}$



(b) O_3

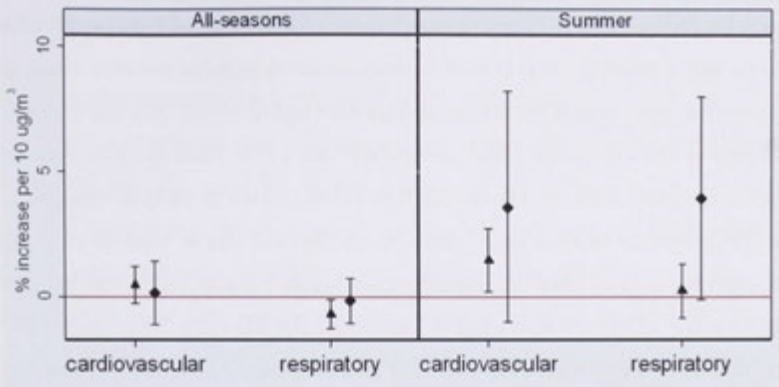


Note: (a) per 10 $\mu g/m^3$ increase in $PM_{2.5}$ and (b) per 10 ppb increase in O_3

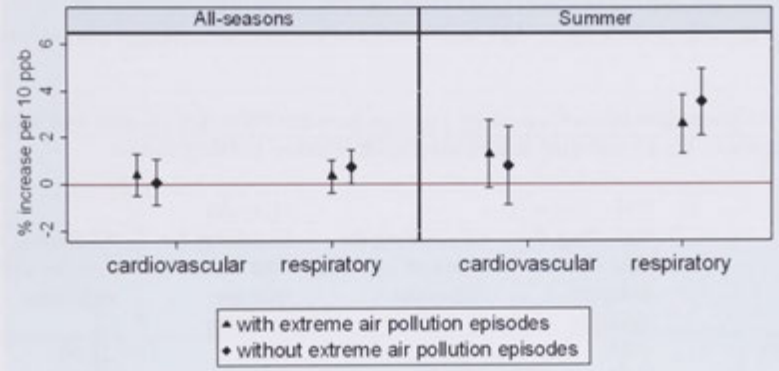
Figure 5.11 Estimates of percentage changes (95% CI) in mortality in the all-ages group derived from using the standard approach and the single-pollutant model with and without air pollution readings on bushfire and dust storm days

ED visits risk estimates associated with $PM_{2.5}$ and O_3

(a) $PM_{2.5}$



(b) O_3



Note: (a) per 10 $\mu g/m^3$ increase for $PM_{2.5}$ and (b) per 10 ppb increase O_3

Figure 5.12 Estimates of percentage changes (95% CI) in ED visits in the all-ages groups derived from using the standard approach and the single-pollutant model with and without air pollution readings on bushfire and dust storm days

The significant positive associations between mortality and air pollution found based on the re-analysis may result from an increase in variance of ambient air pollution exposures, particularly for $PM_{2.5}$. Table 5.3 presents coefficients of variations of $PM_{2.5}$ and O_3 based on data with and without the air pollution measurements on bushfire and dust storm days. It can be seen that removing the readings on days with bushfires and dust storms had a greater impact on the variance of $PM_{2.5}$ compared to that of O_3. Thus, the size, significance and CI of the mortality risks associated with $PM_{2.5}$ based on the re-analysis changed to a greater extent compared to those associated with O_3. The increase in variance of exposure to air pollution not only affected the mortality association, but also influenced the power of detecting the effect of air pollution on daily cardiovascular ED visits. This finding is probably related to a small number of events of cardiovascular ED visits, compared to those of respiratory ED visits. This can be seen from the stronger or significant positive associations between cardiovascular ED visits and $PM_{2.5}$ exposures based on the re-analysis.

Table 5.3 Comparison of coefficients of variation between $PM_{2.5}$ and O_3 with and without the inclusion of the air pollution data on days with bushfire and dust storms

	$PM_{2.5}$ ($\mu g/m^3$)		O_3 (ppb)	
	Including the data on extreme episodes	Excluding the data on extreme episodes	Including the data on extreme episodes	Excluding the data on extreme episodes
Mean	7.42	7.08	23.21	21.90
SD	6.64	4.14	9.25	8.94
Coefficient of variation (%)	89.49	58.47	39.85	40.82

5.3.2 Non-linear model assumption

To explore whether or not the relationship between mortality and exposure to $PM_{2.5}$ in the Melbourne Region is linear, Model 5.1 was refitted by using the data with bushfire and dust storm days and by changing the term $pollutant_{t,0-2}$ for $PM_{2.5}$ from linear to quadratic. Based on the results found in the previous section, which demonstrate a positive significant association between $PM_{2.5}$ and cardiovascular mortality, the exploration in this section focused only on cardiovascular mortality. Figure 5.13 shows that a non-linear relationship between cardiovascular mortality and exposure to ambient $PM_{2.5}$ could tentatively be present. Seemingly, the quadratic curve approximates two linear dose-response functions in which the transition in their slopes appears to lie at the PM

concentrations between 40 to 60 $\mu g/m^3$. Note that these concentrations are consistent with a $PM_{2.5}$ maximum concentration of 60 $\mu g/m^3$ when the $PM_{2.5}$ data on days with the extreme air pollution events were removed.

Although the plot may indicate a non-linear relationship, a likelihood ratio (LR) test failed to reject the null hypothesis that there was no curvature in the association between cardiovascular mortality and $PM_{2.5}$ exposure at the p-values of 0.10 and 0.51 for the model analysing all seasons and the summer respectively. This suggests that there is little evidence to support the nonlinear-relationship. It is possible that any non-linear relationship may reflect the variation in dose-response functions among different components of $PM_{2.5}$, and the test performed here simply failed to detect the non-linearity. Research on this issue is currently active and more information gained from the growing body of research, particularly from North America, may further provide more insight into the relationship between the chemical components of $PM_{2.5}$ and health effects (Bell 2012).

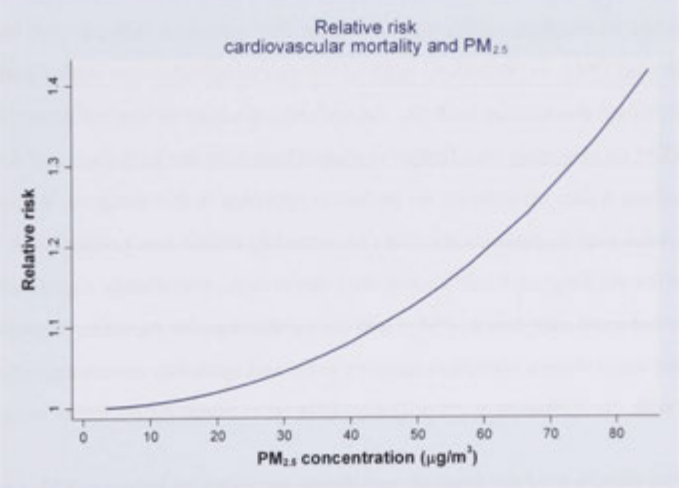


Figure 5.13 A quadratic relationship between cardiovascular mortality and exposure to $PM_{2.5}$ in all seasons and the summer

5.4 Chapter summary

In this chapter, day-to-day variations in health and air pollution data with adjustment of weather parameters were modelled to estimate risks of mortality and ED visits attributable to $PM_{2.5}$ and O_3 exposure in the Melbourne Region. Based on the observed and blended datasets of air pollution and weather described in Chapters 3 and 4, four approaches, including standard, SLA, nearest and blending, were compared to identify which was the best at estimating health risks that could be used further in assessing the impact of climate change on the health effects of air pollution. The blending approach was identified to be the best approach as it provided larger health risk estimates for $PM_{2.5}$ and O_3 than the others. The superiority of the blending approach was largely explained by reduced measurement error in the blended dataset as a result of incorporating intra-urban variation of exposures that may be more suitable when applying it to a large city like Melbourne.

When the blending approach was applied to estimate associations between the concerned health outcomes and exposure to $PM_{2.5}$ and O_3, a significant effect was found in most ED visits outcomes. In contrast, only a very few mortality outcomes had positive significant associations with $PM_{2.5}$ exposure and none of the mortality outcomes investigated had a positive significant association with O_3. An underlying cause of limited detection of the air pollution effect on mortality was further explored based on the hypothesis of the removal of the air pollution data on extreme air pollution episodes in this analysis. When the statistical model used to estimate the risks of mortality earlier was refitted with the inclusion of the readings on bushfire and dust storm days, statistically significant associations between exposure to $PM_{2.5}$ and the cardiovascular mortality became apparent. However, the associations between exposure to O_3 and mortality outcomes remained unchanged with the inclusion of air pollution data on extreme air pollution events.

Following the detection of the positive significant associations between $PM_{2.5}$ and cardiovascular mortality, a quadratic model was fitted to explore whether a non-linear relationship existed based on the hypothesis of the difference in $PM_{2.5}$ composition between normal days and extreme air pollution episode days. A LR test did not confirm that there was significant deviation from linearity in the relationship between exposure to total $PM_{2.5}$ mass and mortality outcomes. Nonetheless, the appearance of a non-linear curve in the fitted quadratic model suggested that a non-linear association between mortality and $PM_{2.5}$ exposure was possible. The hypothesis of the non-linear relationship

should not yet be ruled out, even though there was insufficient evidence in the current analyses.

Based on the comparisons of results from the four approaches, which suggested that blending was superior in terms of detecting the health risks of air pollution, the blending approach will be applied further in the following chapter to detect and estimate the interaction effects of temperature and air pollution on health.

Chapter 6 Interaction between temperature and air pollution on the health effects of air pollution

6.1 Introduction

In the light of the current understanding that temperature and air pollution levels are both likely to alter due to climate change as introduced in Chapter 1, it is important to consider whether and to what extent these two environmental factors may have interacting impacts on human health. As reviewed in Chapter 2, the evidence from past studies suggests that the association between air pollution and health can potentially be modified by temperature. This chapter, hence, aims to achieve Objective 2 of this thesis by using the selected approach presented in Chapter 5 to further investigate whether temperature modifies the effect of air pollution on mortality and morbidity outcomes in the Melbourne Region.

Similar to Chapter 5, the health, air pollution and weather data will be linked to examine changes in the effect of air pollution on health across the temperature range. To quantify relative health risks of air pollution across the temperature range, the stratification approach is used, as has been adopted in several past studies (reviewed in Chapter 2). Since several aspects of model specification for investigating the effect modification may influence the relative risk estimates, sensitivity analyses are also reported to demonstrate robustness of the methods and results. To link with Component 2 of this thesis, this chapter also briefly introduces how the results from this chapter will be applied to assess health impacts of future air pollution in relation to climate change in Chapter 8.

6.2 Statistical methods

Among the statistical methods applied to investigate the modification effect of temperature on the association between air pollution and health as reviewed in Chapter 2, stratification approaches have been widely adopted in many studies. One key advantage of the stratification approach is that it allows quantification of the relative health risks of air pollution across the temperature range. The stratification approach was adopted in this thesis because the ultimate aim was to obtain quantified risk estimates, which might vary in different temperature strata. This then allows the estimations of predicted acute health effects of air pollution due to future changes in air quality related to climate change.

6.2.1 Justifications for the scope of the analyses

Prior to stating the details of the statistical methods applied to investigate the interaction effects in this section, a decision on three matters—i) making further analyses by season or

using only all-seasons data, ii) making a choice between the single- or two-pollutant model to be fitted in the stratification model and iii) proceeding with all-ages or age-group specific analyses—was made to narrow down the scope of the analyses.

The first to be decided was related to whether or not the interaction effects should be specifically considered by season as presented in the previous analyses. Evidence from a large body of studies is suggestive of seasonal variation for the health effects of air pollution (Ito et al. 2007; Chen et al. 2013; Bell et al. 2008; Peng et al. 2005). This is mainly because ambient levels of some air pollutants, in particular O_3 and PM, vary greatly by season due to changes in the sources of air pollution and meteorological conditions. Such variation is highly likely to influence the health effects investigated. Also, the results from the analyses presented earlier in Chapter 5 indicate that the risk estimates associated with air pollution in the Melbourne Region varied considerably by season. Therefore, only a seasonal analysis was continued from this point forward.

In relation to the choice between the single- or two-pollutant models, evidence from studies investigating the health effects from exposure to multiple air pollutants coupled with the health risks estimated by the single- and two-pollutant model presented earlier were used to justify this issue. The results derived from the single- and two-pollutant models, as reported earlier, suggested that i) $PM_{2.5}$ and O_3 may be confounders for the health effects of each other and that ii) O_3 may be acting as a surrogate for $PM_{2.5}$. Tolbert et al. (2007, p. S32-S33) specifically address these two scenarios and provide suggestions on the interpretation of the resulting estimates:

'If the two pollutants are thought to be independent risk factors for the outcome in question and are correlated with each other (positively or negatively), then it may be appropriate to use a two-pollutant model to adjust the effect estimate of each pollutant for confounding by the other pollutant.'

'In a second scenario, if one of the pollutants is etiologically linked to the outcome and the second pollutant is a surrogate for the first pollutant, the two-pollutant model is only useful for determining which pollutant is the better predictor of the outcome. If the etiologic agent is measured with substantially more error than the surrogate (e.g., because of spatial variation or analytic error), measurements of this agent may be less predictive than those of the surrogate, leading to potentially misleading conclusions if the model is interpreted causally.'

According to the suggestions above, on the one hand, if $PM_{2.5}$ and O_3 are considered as confounders to each other, it is recommended to use the risk estimates from the two-pollutant model. On the other hand, if O_3 is considered to be a surrogate to $PM_{2.5}$, only the risk estimates derived from the single-pollutant model of O_3 should be used further.

Two studies by Sarnat et al. (Sarnat et al. 2001; Sarnat et al. 2005) may also be relevant. The studies suggested that if there is a possibility that the concentration of a gaseous pollutant is a surrogate for levels of PM pollutants, then the relative risk from a single-pollutant model should be used. This suggestion was based on the fact that a relative risk derived from a multiple pollutant model is likely to be underestimated for a pollutant that is less predictive and overestimated for a surrogate pollutant. Hence, it is recommended to use a risk estimate from a single-pollutant regression model.

Due to a wealth of evidence of $PM_{2.5}$ and O_3 acting as confounders of each other for health effects plus avoidance of the problem of over- and under-estimation yielded from the two-pollutant model, the analyses in this chapter followed the recommendation made by Sarnat et al. (Sarnat et al. 2001; Sarnat et al. 2005). Therefore, a stratification model with only single pollutants was applied in the analyses of the possible interaction effects.

Although the risk estimates associated with $PM_{2.5}$ and O_3 presented in Chapter 5 were apparently different across the age groups, in that children and the elderly were the most susceptible, it was inconclusive that the relative risks estimated specifically for these age groups were statistically different. To make a decision on this issue, Z and Wald tests were performed when age was classified into two and three groups respectively. The tests examined the equality of the risk estimates associated with $PM_{2.5}$ and O_3 between the different age groups in the single-pollutant model in the summer and the winter, as previously presented in Chapter 5. Testing of the results (see Appendix D.1) suggested that there was no statistical difference in relative risks among the different age groups. Hence, the analysis presented from this point forward focused only on the health risk with no age classifications.

6.2.2 Stratification model

The model below, which is referred to as the 'stratification model', was modified from Model 5.3 as explained in Chapter 5, and is referred to here as the 'basic model'. To investigate the modification effect of temperature on the association between air pollution and health, temperatures were stratified. The parameter of temperature strata was entered into the stratification model as an interaction term with the air pollutant parameter.

$$log\left(\mu_{i,t}\right) = \alpha + \sum_{k=1}^{n} \beta_k \, pollutant_{i,t_{,0-2}} I(tempstrata = k) + \sum_{i=1}^{N} \beta_2 \, (time) I(sla = i) + s_1\left(avgtemp_{i,t}, 6df\right) + s_2\left(avgtemp_{i,t,1-3}, 6df\right) + s_3\left(avgdew_{i,t}, 4df\right) + s_4\left(avgdew_{i,t,1-3}, 4df\right) + s_5(time, 7df \; per \; year) + \boldsymbol{\gamma_1 DOW} + \boldsymbol{\gamma_2 influenza} + \boldsymbol{\gamma_3 holiday} + \sum_{i=1}^{N} \boldsymbol{\gamma_{4,i}} \boldsymbol{I(sla = i)} \quad (6.1)$$

In Model 6.1, the previous main effect of air pollution (β_1) in the basic model was replaced by an interaction term between the main effect of the pollutants of concern (β_k) and temperature strata ($tempstrata$). Stratifying temperature was based on percentiles of the mean of maximum dry-bulb temperature on day *t* and minimum dry-bulb temperature on day *t-1* or $avgtemp_t$.

6.2.3 Identification of temperature cut-points

The goal here was to identify the best cut-point(s) across the disease categories (cardiovascular and respiratory diseases), the health endpoints (mortality and ED visits) and the pollutants ($PM_{2.5}$ and O_3) for each season that could be used in stratifying the temperature. This means that there were eventually only two sets of cut-point(s)—one for the summer and the other for the winter—used to estimate changes in relative health risks across the temperature range. No analysis was conducted to explore the health effects of O_3 exposure in the winter. Therefore, the search for the best cut-point(s) in the winter was done only for $PM_{2.5}$. The analysis was limited to cardiovascular and respiratory disease, not all-cause mortality.

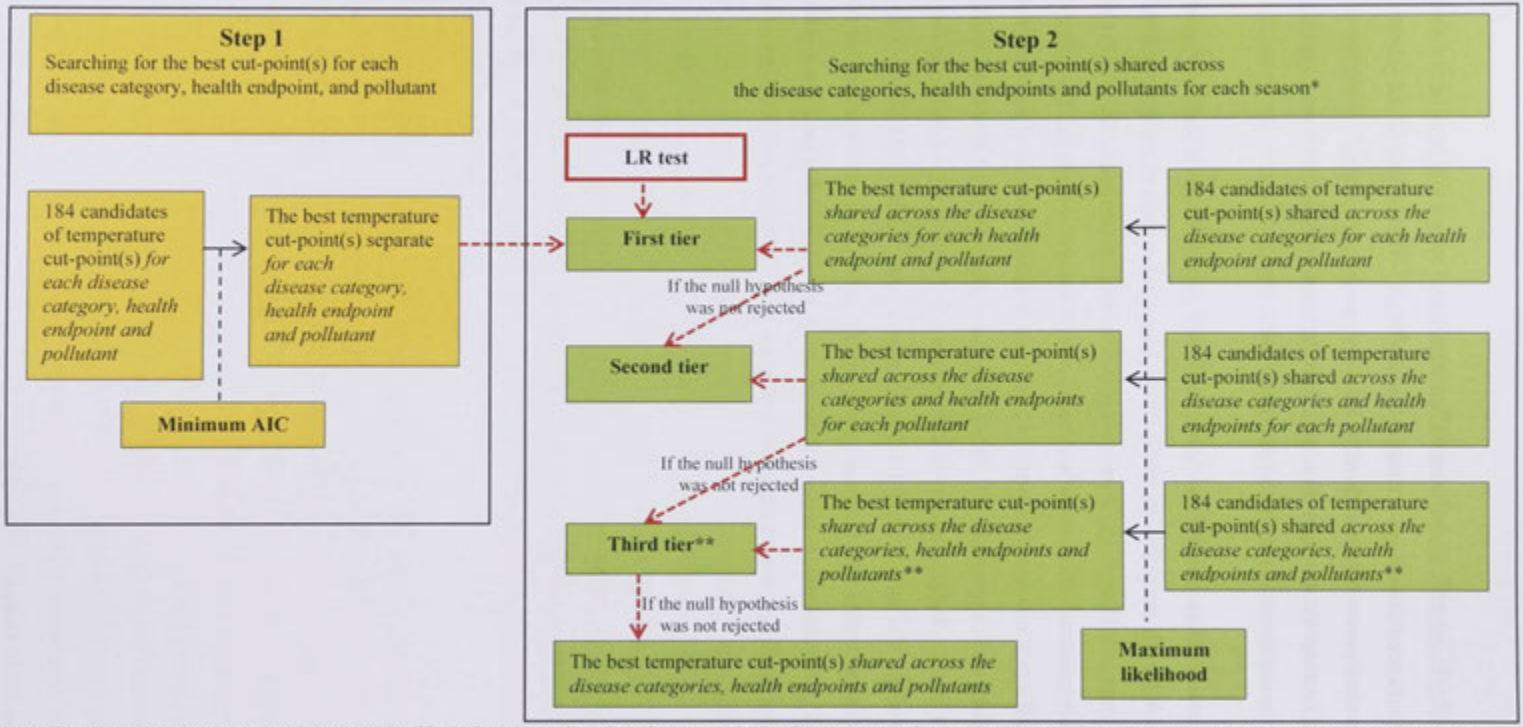
To achieve the goal, two steps were taken to identify the best temperature cut-point(s) as shown in Figure 6.1. Step 1 involved searching for the best cut-point(s) for each disease category, health endpoint, pollutant and season. After these candidate cut-points were identified, a search was conducted again in Step 2 to acquire the same cut-point(s) shared among the disease categories, the health endpoints and the pollutants for each season.

In Step 1, the first thing to be considered was how many cut-points should be used to stratify the temperatures. Here, the strategy of temperature stratification was rationalised on the basis of the knowledge of the shapes of temperature-health relationships. As reviewed in Chapter 2, this strategy was also adopted in one study investigating the temperature modifying effect on the health risk associated with O_3 levels (Pattenden et al. 2010). Evidence from past studies suggests that the temperature-health relationship usually falls into one of three shapes, namely a U- J- or V-shape (Turner et al. 2012; Ye et al. 2012; Yu et al. 2012; Basu 2009). For all three shapes, one or two temperature threshold values are identified. Between or at the threshold value(s), it is generally apparent that the health impact associated with exposure to temperature is minimal. With the minimal health impact from temperature, the human body is likely to have a low level of physiological stress, resulting in a higher tolerance level to other health risks including air pollutants. In contrast when the human body is exposed to temperature outside the comfort zone (outside the threshold values), this could raise the stress level and may consequently lead to higher susceptibility of the body to other hazards. Building on this knowledge, this analysis explored two alternatives: the use of a single temperature cut-point or a pair of cut-points in stratifying the temperatures to explore the modification effect of temperature on the health effects of air pollution.

Choosing the temperature cut-points in Step 1 was based on AIC values obtained from fitting Model 6.1. For a given air pollutant, disease category and health endpoint, a single cut-point or a pair of cut-points was selected that gave the lowest AIC. In other words, a single cut-point or a pair of cut-points that was ranked first among all of the candidate cut-points on the basis of the minimum AIC value was selected. The temperatures were divided into every 5^{th} percentile with the percentile cut-points considered starting from the 5^{th} to 95^{th} percentiles. The upper limit of the single cut-points was at the 80^{th} percentile. For the pairs of cut-points, the 80^{th} percentile was the lower bound of the highest pair. In total, there were 184 sets of temperature cut-points in the percentiles considered, of which there were 16 single cut-points (5^{th}, 10^{th},..., 80^{th} percentiles) and 168 pairs of cut-points

(5^{th} and 10^{th}, 5^{th} and 15^{th},..., 80^{th} and 95^{th} percentiles). Using a single cut-point divided the temperature percentiles into two strata—low and high—while a pair of cut-points divided the temperature percentiles into three strata—low, middle and high. A Wald test was performed to examine the equality of risk estimates between the different temperature strata for the cut-point(s) giving the minimum AIC.

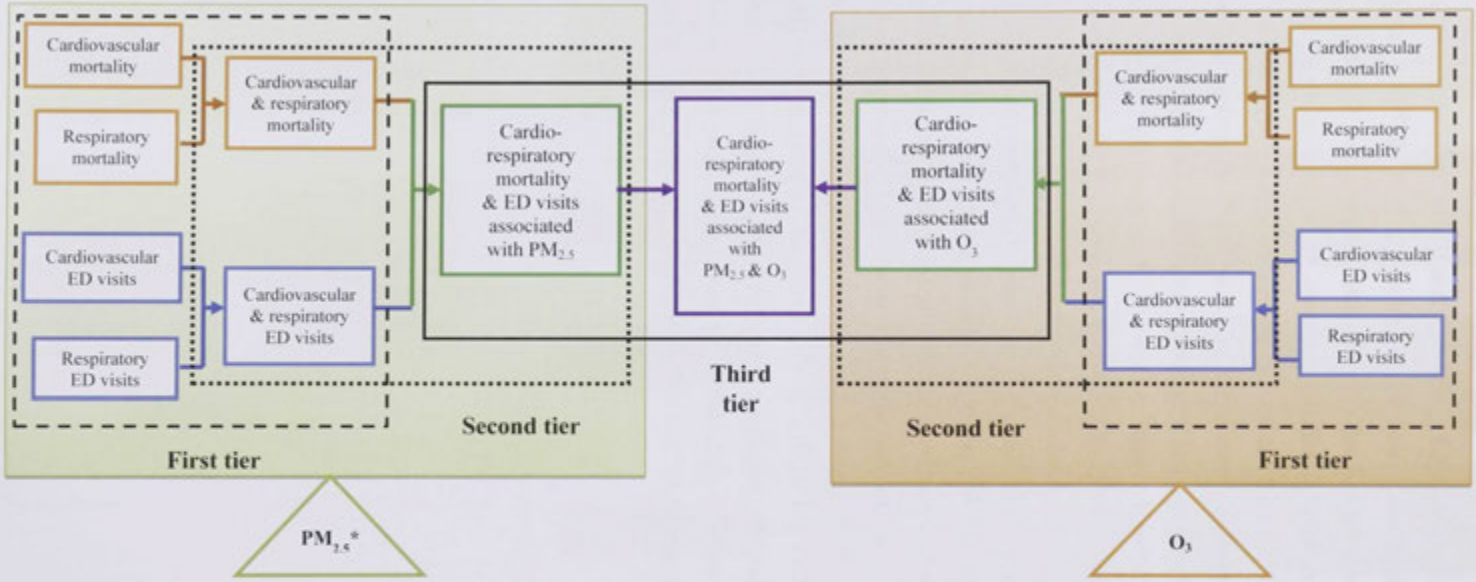
In Step 2, the best cut-point(s) was identified based on results of LR tests. As reported later in Section 6.3.1, all cut-points identified from Step 1 were pairs of cut-points. This indicates that overall, applying a pair of cut-points was best-suited to the context of investigating possible effect modification by temperature in this thesis. Therefore, the search for the best cut-points in Step 2 considered only a pair of cut-points identified from Step 1, which gave a minimal AIC for each disease category. The LR tests in Step 2 were conducted in a tier fashion, as depicted in Figure 6.2. LR tests were conducted for three and two tiers for the summer and the winter respectively. A LR test for the first tier was undertaken to examine whether or not two separate pairs of cut-points identified from Step 1—one for cardiovascular disease and the other for respiratory disease—for a given health endpoint and pollutant were better than a single pair of cut-points applied across these two disease categories.



Note: *In winter, the search was conducted to identify the best cut-points shared across only the disease categories and health endpoints. This is because the analysis was conducted to explore only the health effects of $PM_{2.5}$. **The third tier was conducted only for summer. *AIC* Akaike's Information Criteria, *LR* Likelihood Ratio.

Figure 6.1 Flow diagram of Step 1 and Step 2 in identifying the best temperature cut-point(s) shared across disease categories, the health endpoints and the pollutants, for each season, used in stratifying the temperatures into different strata

Table 6.1 gives an example of the LR test for this first tier for $PM_{2.5}$ associated with mortality in the summer. It can be seen that the cut-points of 70^{th}, 85^{th} percentiles and 35^{th}, 95^{th} percentiles were the best pairs of cut-points identified from Step 1 for cardiovascular and respiratory mortality respectively. These two pairs were compared with another pair of cut-points, which was the best pair identified from Step 2 for both cardiovascular and respiratory diseases. The pair of 40^{th}, 50^{th} percentiles was identified as the best one of the 184 candidates of temperature cut-points, which gave the maximum likelihood (ML) from fitting the stratification model. To perform the LR test in this example, a difference between ML-A and ML-B was computed. ML-A was a product of adding two MLs, one each for cardiovascular and for respiratory disease, derived from fitting the stratification model using the best two pairs of cut-points (the cut-points of 70^{th}, 85^{th} percentiles and 35^{th}, 95^{th} percentiles) identified from Step 1. ML-B was a product of adding two MLs, each for cardiovascular and for respiratory disease, based from fitting the stratification model using the best single pair of cut-points (the cut-points of 40^{th} and 50^{th} percentiles) identified from Step 2. A significance test was conducted to examine the difference between ML-A and ML-B at p-value 0.05 level. In the example in Table 6.1, the p-value was greater than 0.05, suggesting the null hypothesis of no difference between ML-A and ML-B cannot be rejected. Hence, the parsimonious model with the single pair of cut-points (the cut-points of 40^{th} and 50^{th} percentiles) identified in Step 2 was chosen.



Note: *In the winter, performing LR tests consists of only two tires for $PM_{2.5}$.
The first tier includes the boxes surrounded by the dashed line. The second tier includes the boxes surrounded by the dotted line. The third tier includes the boxes surrounded by the solid line.

Figure 6.2 Conducting LR tests in Step 2 to identify the best pair of temperature cut-points across the disease categories, the health endpoints and pollutants.

Table 6.1 An example of performing the LR test in the first tier of Step 2 for $PM_{2.5}$ associated with mortality in the summer

	A* each pair of cut-points per disease category		B** each pair of cut-points per health endpoint		ML-A minus ML-B	p-value of LR test
	Cut-points in percentiles	ML-A	Cut-points in percentiles	ML-B		
CVD	70, 85	-27537.26	40, 50	-27540.24		
		+		+		
RD	35, 95	-9873.17	40, 50	-9875.51		
		-37410.43		-37415.75	5.32	0.07

Note: A* A pair of temperature cut-points was applied for each disease category, health endpoint and pollutant; B** A pair of temperature cut-points was applied for each health endpoint and pollutant; *CVD* cardiovascular disease, *RD* respiratory disease

In the first tier of Step 2, if a single pair of cut-points suiting both cardiovascular and respiratory disease was proved to be superior to two pairs of cut-points suiting each of the disease categories, the single pair of cut-points identified from this tier was tested further in the second tier, as illustrated in Figure 6.1. In the second tier, the same process using the LR test was repeated again. However, this time it was used to compare MLs of two separate pairs of cut-points identified from the first tier—one for cardiorespiratory disease mortality and the other for ED visits—for a given pollutant with MLs of a single pair of cut-points applied across these health endpoints identified from the second tier. The analyses for the winter ceased at the second tier while those for summer proceeded to the third tier. This was because no analysis was conducted to explore the O_3 effects in the winter. In the third tier, the LR test was used to compare MLs of two separate pairs of cut-points identified from the second tier—one for cardiorespiratory disease mortality and ED visits associated with $PM_{2.5}$ and the other for O_3—in the summer with MLs of a single pair of cut-points applied across $PM_{2.5}$ and O_3 identified from the third tier.

Note that if the null hypothesis of the LR test in the first or second tiers was rejected, the LR test had to be concluded and the continuation to the next tier would not proceed. This would mean that the two pairs of cut-points were superior to the single pair of cut-points and should be used to fit Model 6.1 at the end of Step 2. However, as reported later in Section 6.3.2, there was only one LR test—comparing two pairs of cut-points, for O_3 associated with cardiovascular and respiratory morality in the summer, with a single pair of cut-points across both disease categories—at the first tier, where the null hypothesis was rejected. Nevertheless, overall the use of a single pair of cut-points across all of the disease categories, health endpoints and pollutants, specific to each season, was not significantly different to using cut-points that were separately identified. Therefore, Model 6.1 was fitted using a single pair of cut-points across the disease categories, the health endpoints and the pollutants for the summer at the third tier and another single pair of cut-points across the disease categories and the health endpoints for the winter at the second tier.

After identifying a single pair of cut-points for each season at the second tier for the winter and the third tier for the summer, a Wald test was performed to test the equality of risk estimates generated by fitting Model 6.1 between the different temperature strata. The null hypothesis is that the estimates across the temperature strata are not different. If the null hypothesis was not rejected, this indicated that there was no evidence of an interaction effect. In contrast, if the null hypothesis was rejected, it indicated the existence of an interaction effect.

6.3 Results

6.3.1 Identified temperature cut-points derived from Step 1

In searching for the best pair of cut-points in Step 1, results of the identified cut-points as reported in Table 6.2 suggested that the pairs of cut-points provided better fit in the stratification model than the single cut-points. It can be seen that in no case does any single cut-point, when used to fit the stratification model, give the minimum AIC for any of the air pollutants and health effects investigated.

There was one consistent pattern observed from the cut-points identified for the same disease category for a given pollutant in the summer. That is, there was a higher percentile of upper cut-point for mortality compared to ED visits. For example, the upper cut-point identified for the association between $PM_{2.5}$ and cardiovascular mortality was the 85th percentile; higher than that identified for the association between $PM_{2.5}$ and cardiovascular ED visits which was the 80th percentile. This pattern probably reflects that mortality is the most severe form of adverse health effect, compared to other health endpoints, including ED visits. Therefore, at a given pollutant concentration, an effect on mortality requires exposure to a higher temperature than an effect on ED visits.

In the winter, one might expect to see a pattern in which the lower cut-point identified for an association with mortality would be more extreme, i.e., lower, than that for ED visits. This is based on the assumption that at a given pollutant concentration, the development of the effect of air pollution on mortality would require exposure to a colder temperature than that for ED visits. However, the pattern found here was the reverse. For cardiovascular disease, the percentile of lower cut-points identified for ED visits was slightly lower than that of mortality. This atypical result may be related to the moderate cold in the winter in Melbourne, which may not be sufficient to cause a distinguishable result of the lower cut-point between mortality and ED visits. For mortality due to respiratory disease, the Wald test does not indicate a difference in the $PM_{2.5}$ effect across the temperature range. Thus, although the estimated percentile of the lower cut-points for ED visits associated with respiratory disease was substantially lower than those for mortality, no reliance can be placed on this finding.

Table 6.2 Identified temperature cut-points based on AIC in Step 1 and results of Wald tests specific to each disease category, health endpoint and pollutant, according to season

Pollutant	Health endpoint	Disease category	Best cut-points in percentiles (in °C)	p-value of Wald test*
Summer				
$PM_{2.5}$	Mortality	CVD	70,85 (21.5, 24.0)	0.05
$PM_{2.5}$	ED visits	CVD	70,80 (21.4, 22.9)	0.02
$PM_{2.5}$	Mortality	RD	35,95 (17.8, 26.2)	0.08
$PM_{2.5}$	ED visits	RD	15,50 (15.8, 19.1)	<0.01
O_3	Mortality	CVD	30,85 (17.3, 24.0)	0.04
O_3	ED visits	CVD	15,70 (15.8, 21.4)	<0.01
O_3	Mortality	RD	40,90 (18.2, 24.9)	0.14
O_3	ED visits	RD	60,75 (20.2, 22.0)	<0.01
Winter				
$PM_{2.5}$	Mortality	CVD	25,75 (8.9, 11.2)	0.02
$PM_{2.5}$	ED visits	CVD	15,50 (8.3, 10.0)	0.04
$PM_{2.5}$	Mortality	RD	75,85 (11.2, 11.9)	0.14
$PM_{2.5}$	ED visits	RD	15,40 (8.3, 9.6)	<0.01

Note: * Testing the equality of coefficients at the different temperature strata (df=2)
CVD cardiovascular disease, *RD* respiratory disease

In general, results of the Wald tests as indicated by the p-values less than 0.05 presented in Table 6.2 were suggestive of the existence of a temperature modifying effect on the health impacts associated with air pollution analysed in this thesis. There were three exceptions in which the results from the Wald tests indicated that the modifying effect did not exist. These included the $PM_{2.5}$ and O_3 effects associated with respiratory mortality in the summer and the $PM_{2.5}$ effect associated with respiratory mortality in the winter. It was apparent that all these effects held one thing in common, which was when respiratory mortality was the health outcome of interest.

6.3.2 Identified temperature cut-points derived from Step 2 and the existence of effect modification

Results of identified cut-points in each tier of Step 2 with corresponding p-values of LR testing are presented in Table 6.3. Except for the p-value of the LR test in the first tier for the association of O_3 with cardiovascular and respiratory morality in the summer, which was less than 0.05, the other p-values of LR tests used to compare the cut-points in each tier were greater than 0.05. This indicated that the use of a single pair of cut-points across all the disease categories, health endpoints and pollutants in the summer identified in Step 2, resulted in models that were not significantly different from those using cut-points specific for disease categories, health endpoints and pollutants identified in Step 1.

Similarly for the winter, this suggested that there was no statistical difference between the use of a single pair of cut-points across all the disease categories and health endpoints for $PM_{2.5}$ and the use of specific cut-points for disease categories and health endpoints. The pair of cut-points of the 15th and 70th percentiles, which corresponded to the temperatures at 15.9 °C and 21.5°C, was identified as providing the best fitting stratification model for the summer. For winter, the best pair of cut-points was the 15th and 85th percentiles, which corresponded to the temperatures at 8.4 °C and 11.9°C.

Table 6.4 shows the p-values of the Wald tests used to examine whether there was a significant interaction between ambient pollutants and temperature on the health outcomes, using the single pair of cut-points identified across all the disease categories, health endpoints and pollutants, for each season, to fit the stratification model. Figure 6.3 shows estimates of percentage changes and 95% CI in each disease category, health endpoint, pollutant and season in the different temperature strata stratified by these two best identified pairs of cut-points (see the estimates presented in tabular form in Appendix D.2). In general, using the single pair of cut-points for each season, a temperature modifying effect was detected in the associations between the air pollutants and ED visits, i.e., the p-values of the Wald test for the ED visits outcomes were mainly less than 0.05. For the $PM_{2.5}$-related cardiovascular ED visits in the summer and in the winter, the p-values were of borderline significance. All p-values of Wald tests for the associations between mortality and $PM_{2.5}$ and O_3 were greater than 0.05. This suggested that there was little evidence that temperature modified the effects of the air pollutants on either cardiovascular or respiratory mortality.

Table 6.3 The best identified cut-points across the disease categories, health endpoints and pollutants based on LR tests in Step 2 and p-values of LR tests, presented separately by season

Pollutant	Health endpoint	Disease category	Cut-points[a,c]	First tier[b]		Second tier[c]		Third tier[d]	
				p-value of LR test	Identified cut-points[e]	p-value of LR test	Identified cut-points[e]	p-value of LR test	Identified cut-points[e]
Summer									
$PM_{2.5}$	Mortality	CVD	70, 85(21.5, 24.0)	0.07	40, 50 (18.2, 19.2)	1.00	15, 95	0.35	15, 70
$PM_{2.5}$	Mortality	RD	35, 95(17.8, 26.2)				(15.8, 26.1)		(15.9, 21.5)
$PM_{2.5}$	ED visits	CVD	70, 80(21.4, 22.9)	0.56	15, 90 (15.8, 24.8)				
$PM_{2.5}$	ED visits	RD	15, 50(15.8, 19.1)						
O_3	Mortality	CVD	30, 85(17.3, 24.0)	0.04	15, 85 (15.9, 24.0)	0.51	15, 70		
O_3	Mortality	RD	40, 90(18.2, 24.9)				(15.8, 21.4)		
O_3	ED visits	CVD	15, 70(15.8, 21.4)	0.28	15, 70 (15.8, 21.4)				
O_3	ED visits	RD	60, 75(20.2, 22.0)						
Winter									
$PM_{2.5}$	Mortality	CVD	25, 75 (8.9, 11.2)	0.63	75, 85 (11.2, 11.9)	0.19	15, 85	NA	NA
$PM_{2.5}$	Mortality	RD	75, 85(11.2, 11.9)				(8.4, 11.9)		
$PM_{2.5}$	ED visits	CVD	15, 50 (8.3, 10.0)	0.87	5, 15 (7.5, 8.3)				
$PM_{2.5}$	ED visits	RD	15, 40 (8.3, 9.6)						

Note: [a] Each pair of cut-points presented in each row of this column was identified for each disease category, health endpoint and air pollutant; [b] The first tier tested the same cut-points across the disease categories for each health endpoint and pollutant; [c] The second tier tested the same cut-points across the disease categories and the health endpoints for each pollutant; [d] The third tier tested the same cut-points across the disease categories, health endpoints and pollutants; [e] All the temperature cut-points presented in this table were expressed in percentiles with the unit of ˚C in the parentheses; *CVD* cardiovascular disease, *RD* respiratory disease

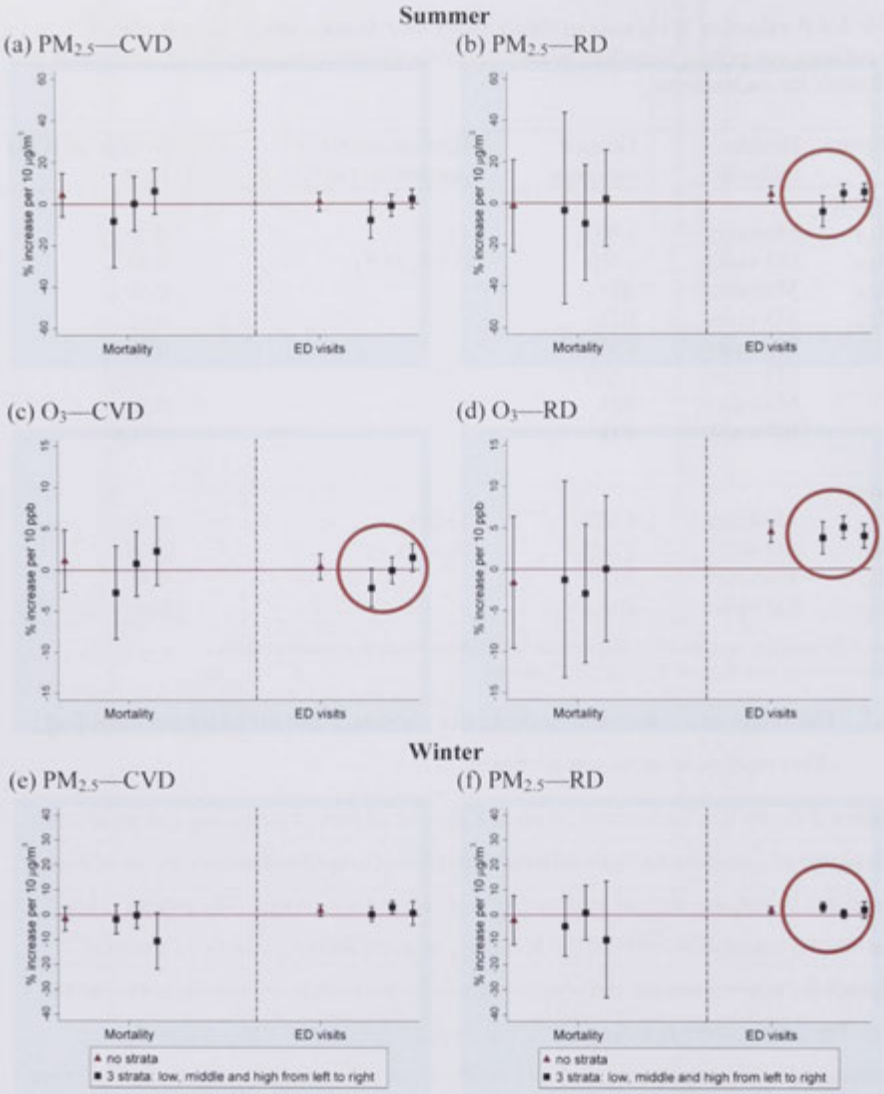
Table 6.4 P-values of Wald tests to detect effect modification using the best pair of temperature cut-points, identified in Step 2, across disease categories, health endpoints and pollutants for each season

Pollutant	Health endpoint	Disease category	Best cut-points in percentiles (in °C)	p-value of Wald test*
Summer				
$PM_{2.5}$	Mortality	CVD	15, 70	0.40
$PM_{2.5}$	ED visits	CVD	(15.9, 21.5)	0.07
$PM_{2.5}$	Mortality	RD		0.60
$PM_{2.5}$	ED visits	RD		0.02
O_3	Mortality	CVD		0.18
O_3	ED visits	CVD		<0.01
O_3	Mortality	RD		0.58
O_3	ED visits	RD		<0.01
Winter				
$PM_{2.5}$	Mortality	CVD	15, 85	0.15
$PM_{2.5}$	ED visits	CVD	(8.4, 11.9)	0.06
$PM_{2.5}$	Mortality	RD		0.41
$PM_{2.5}$	ED visits	RD		0.01

Note: * Testing the equality of coefficients at the different temperature strata (df=2)
CVD cardiovascular disease, *RD* respiratory disease

6.3.3 The relationship between acute health outcomes and air pollution including effect modification by temperature

Figure 6.3 shows that for summer, there is a general pattern of increasing risk estimates with higher temperature for both pollutants in relation to cardiovascular outcomes, even though the interaction was not statistically significant for mortality. The pattern is less clear for the association between the pollutants and respiratory outcome in summer, although the interactions are statistically significant for both pollutants in relation to ED visits. For winter, there is a decrease in the association between $PM_{2.5}$ and both cardiovascular and respiratory mortality for the highest temperature stratum only. Except for the association of $PM_{2.5}$ with respiratory ED visits for winter, the interaction for other health outcomes is not statistically significant.



Note: The estimates were derived from the stratification model using the three temperature strata stratified by the cut-points presented in Table 6.4. The estimates marked with no strata were derived from fitting the basic model. The risk estimates circled in red were those where the p-values of the Wald tests indicated that there was a interaction effect.
CVD cardiovascular disease, *RD* respiratory disease

Figure 6.3 Estimates of percentage changes in three temperature strata and 95% CI in each disease category, health endpoint and pollutant presented by season.

There was evidence of statistically significant effect modification by temperature for the effect of air pollution on four health outcomes; these are considered in more detail below. These outcomes—respiratory ED visits associated with $PM_{2.5}$, cardiovascular ED visits associated with O_3, respiratory ED visits associated with O_3 in the summer and respiratory ED visits associated with $PM_{2.5}$ in the winter—are circled in red in Figure 6.3.

The patterns of risk estimates in the three temperature strata for respiratory ED visits associated with $PM_{2.5}$ and for cardiovascular ED visits associated with O_3 in the summer were the same. The strongest associations were found in the upper temperature stratum. For the risk estimates for respiratory ED visits associated with $PM_{2.5}$, the estimate in the upper temperature stratum was statistically significant and was higher than that estimated using the basic model without an interaction term. Estimating the health risks associated with O_3 and cardiovascular ED visits using the stratification model resulted in a positive association in the upper temperature stratum that was of borderline significance. Notably, the estimate from the basic model was of non-significant effect with a magnitude close to the null.

The pattern of the risk estimates across the three temperature strata were different for the association between O_3 and respiratory ED visits compared to the association with cardiovascular ED visits. For respiratory ED visits, the largest estimate of effect was in the middle temperature stratum, followed by the estimates for the upper and lower strata respectively. For cardiovascular ED visits, there was a monotonic increase in the risk estimates with increasing temperature stratum. This different pattern may be the result of other underlying determinants that were not identified here.

For the winter, among the three temperature strata, the highest estimate of associations between respiratory and ED visits and $PM_{2.5}$ was found in the lower temperature stratum. This estimate was higher than that estimated by the basic model, which assumed no interaction effects. Likewise a significant risk estimate was found only for the lower temperature stratum.

Overall, the results suggested modification of the associations between the two pollutants and ED visits by temperature. Potentially, the adverse health effects due to exposure to air pollution could be more pronounced on hot days in the summer and on cold days in the winter.

6.3.4 Sensitivity analyses

Sensitivity analyses were conducted to explore the impact of changes in the model specification of two parameters—the lag period of $PM_{2.5}$ and O_3 and the time metric of O_3. The focus of undertaking the sensitivity analyses was to observe the effect of different values for the two parameters on the identified cut-points, and/or the patterns and magnitudes of the associations between the air pollutants and the health effects across the different temperature strata. To test whether the results derived from the stratification model reported above were sensitive to such changes, a lag of 3-day moving average (L02) was replaced with a lag of 2-day moving average (L01) for both pollutants. The main reason to choose L01 here was that it has also been widely used in epidemiological studies of air pollution (USEPA 2009, 2013). Likewise, the max 8-h metric of O_3 was replaced by the max 1-h metric, which is one of the two metric times used in the ambient standards for O_3 in Australia.

Table 6.5 shows that the change in the lag period had a modest impact on the identified pair of cut-points, i.e., the pair of cut-points used to fit Model 6.1 and gave the minimum AIC value, for both pollutants in Step 1 for the selection of the best cut-point(s) for each disease category, health endpoint and pollutant. The highest impact was found in the associations between O_3 and respiratory mortality in the summer and $PM_{2.5}$ and cardiovascular ED visits in the winter. For these two health effects, replacing L02 with L01 changed the pair of cut-points, first identified as the best one based on the criterion of the minimum AIC value (or at the first rank), to the 15th rank (among the 184 candidate cut-point(s)). The change in O_3 metric had little influence on the optimal cut-points. The best pairs of cut-points identified for the association between O_3 and cardiovascular and respiratory ED visits remained unchanged, while those identified for the O_3-attributed cardiovascular and respiratory ED mortality were down from the first rank to the second rank.

Step 2 was insensitive to the changes in the two parameters tested. Five cross-matchings between the changes in these two parameters in the summer and the single change of $PM_{2.5}$ lag time in the winter were tested as shown in Tables 6.6, by using the cut-points reported in Table 6.5. Results in Table 6.6 suggested that regardless of the change in either the $PM_{2.5}$ and O_3 lag period or the O_3 metric, or the coupled change of both parameters, the

identified pair of cut-points in each season remained the same (see detailed results in Appendices D.3 and D.4).

Figures 6.4 to 6.6 show patterns and sizes of the associations between the health outcomes of concern and the pollutants in the three different temperature strata when the stratification model was fitted with the changed $PM_{2.5}$ and O_3 lag time and O_3 metric. It can be seen that the change in $PM_{2.5}$ and O_3 parameters did not affect the pattern of the health effect due to air pollution observed previously. The sizes of risk estimates were slightly altered in a few health outcomes when L02 was replaced by L01 (e.g., in Figures 6.4a and 6.5d). P-values of Wald tests that indicate the existence of the temperature modification effects remained unchanged for all health outcomes (see Table 6.7).

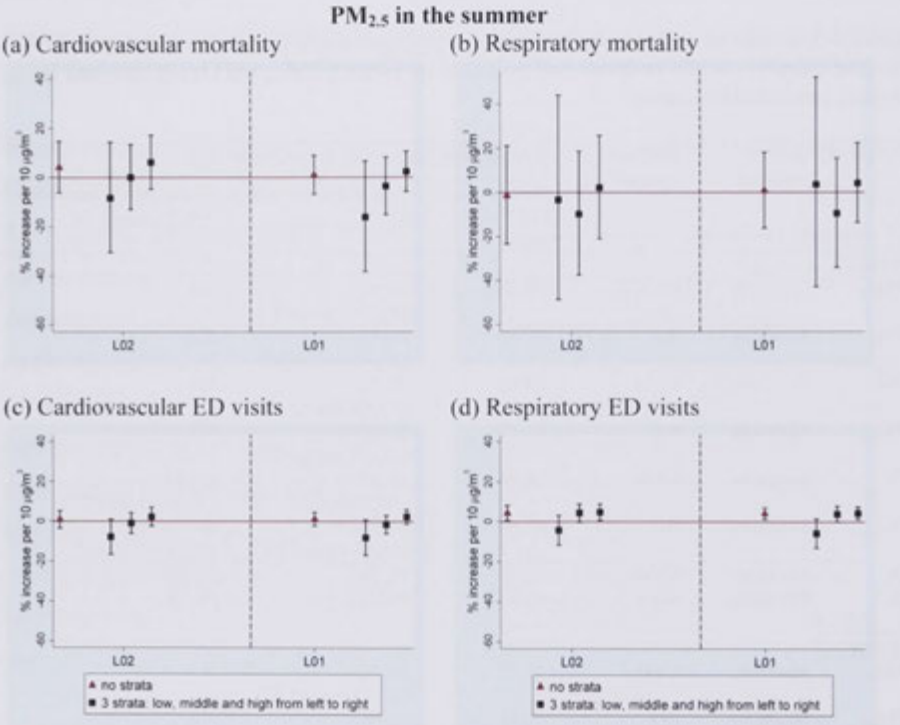
Table 6.5 Results of the best identified cut-points based on AIC in Step 1 specific to each disease category, health endpoint and pollutant by varying $PM_{2.5}$ and O_3 lag time and O_3 metric, presented by season

Pollutant	Health endpoint	Disease category	Best identified cut-points in percentile		
			Benchmark (L02 and O_3 max 8-h)	Replacing L02 with L01*	Replacing O_3 max 8-h with O_3 max 1-h*
Summer					
$PM_{2.5}$	Mortality	CVD	70, 85	70, 95 (70, 85 ranked 4th)	NA
$PM_{2.5}$	Mortality	RD	35, 95	20, 95 (35, 95 ranked 4th)	NA
$PM_{2.5}$	ED visits	CVD	70, 80	15, 25 (70, 80 ranked 10th)	NA
$PM_{2.5}$	ED visits	RD	15, 50	20, 50 (15, 50 ranked 2nd)	NA
O_3	Mortality	CVD	30, 85	15, 85 (30, 85 ranked 2nd)	15, 85 (30, 85 ranked 2nd)
O_3	Mortality	RD	40, 90	60, 90 (40, 90 ranked 15th)	40, 40 (40, 90 ranked 2nd)
O_3	ED visits	CVD	15, 70	15, 70	15, 70
O_3	ED visits	RD	60, 75	60, 75	60, 75
Winter					
$PM_{2.5}$	Mortality	CVD	25, 75	75, 90 (25, 75 ranked 3rd)	NA
$PM_{2.5}$	Mortality	RD	75, 85	75, 85	NA
$PM_{2.5}$	ED visits	CVD	15, 50	5, 15 (15, 50 ranked 15th)	NA
$PM_{2.5}$	ED visits	RD	15, 40	5, 15 (15, 40 ranked 2nd)	NA

Note:*The ranking was based on the criterion of the minimum AIC value as described in Section 6.2.3. The pair of cut-point(s) with the lowest AIC was ranked the 1st. The pair of cut-points in parenthesis was in the first rank (minimum AIC) using the lag period of L02. However the ranking for that pair was changed to the rank indicated in the parenthesis using the lag period of L01 or the O_3 metric of max 1-h.
CVD cardiovascular disease, *RD* respiratory disease

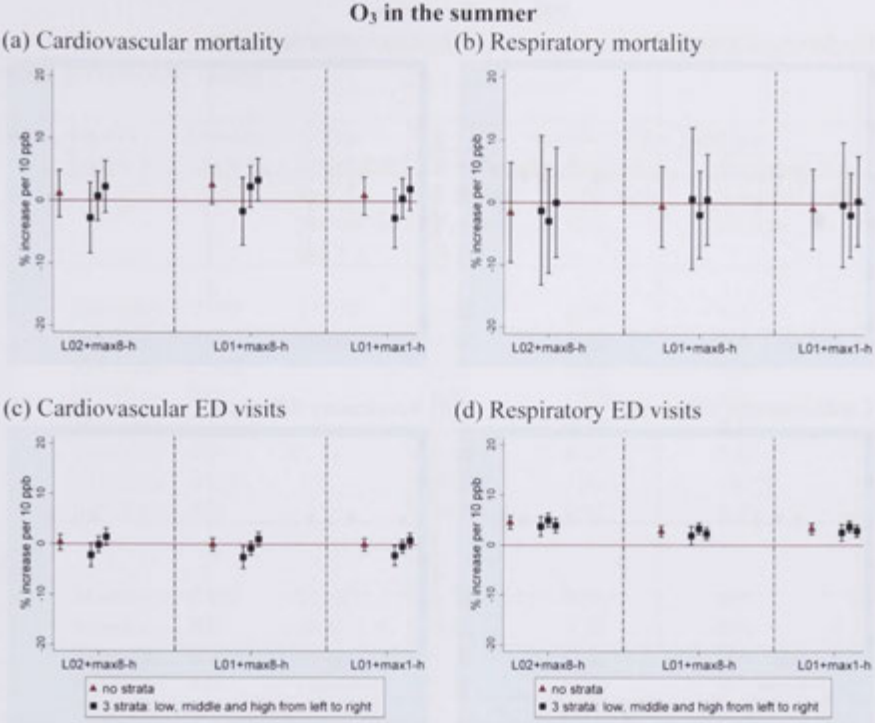
Table 6.6 Results from sensitivity analyses to test the best identified pairs of cut-points based on LR tests in Step 2, across the disease categories, health endpoints, and pollutants, presented by season

Tested parameters	Identified best pair of cut-points in percentile
Summer	
$PM_{2.5}$ L01 + O_3 max 8-h /L01	15,70
$PM_{2.5}$ L01 + O_3 max 1-h /L02	15,70
$PM_{2.5}$ L01 + O_3 max 8-h /L02	15,70
$PM_{2.5}$ L02 + O_3 max 1-h /L02	15,70
$PM_{2.5}$ L02 + O_3 max 8-h /L01	
Winter	
$PM_{2.5}$ L01	15,85



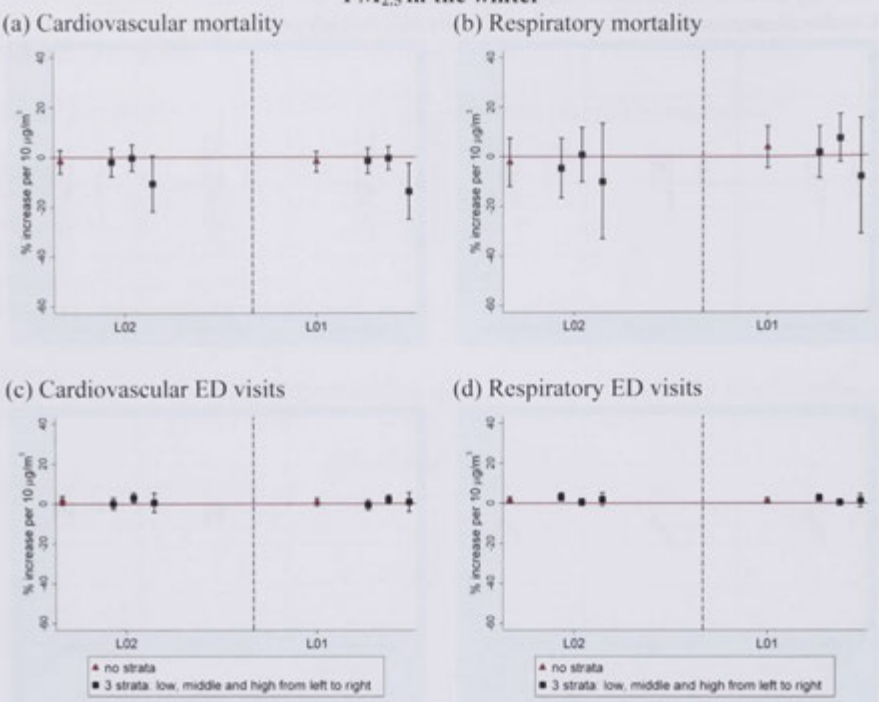
Note: The estimates are presented for three temperature strata—low, middle and high—stratified by using the pair of cut-points of the 15th and 70th percentiles. The estimates marked with no strata were derived from fitting the basic model. The lag period was changed from 3-day moving average (L02) to 2-day moving average (L01).

Figure 6.4 Estimates of percentage changes and 95% CI in each disease category of mortality and ED visits associated with $PM_{2.5}$ per 10 $\mu g/m^3$ in the summer using different lag periods



Note: The estimates are presented for three temperature strata—low, middle and high—stratified by using the pair of cut-points of the 15th and 70th percentiles. The estimates marked with no strata were derived from fitting the basic model. The lag period was changed from 3-day moving average (L02) to 2-day moving average (L01). The O_3 metric was changed from max 8-h to max 1-h.

Figure 6.5 Estimates of percentage changes and 95% CI in each disease category of mortality and ED visits associated with O_3 per 10 ppb in the summer using different lag period and O_3 metrics.



Note: The estimates are presented for three temperature strata—low, middle and high—stratified by using the pair of cut-points of the 15th and 85th percentiles. The estimates marked with no strata were derived from fitting the basic model. The lag period was changed from 3-day moving average (L02) to 2-day moving average (L01)

Figure 6.6 Estimates of percentage changes and 95% CI in each disease category of mortality and ED visits associated with $PM_{2.5}$ per 10 $\mu g/m^3$ in the winter using different lag periods.

Table 6.7 P-values of the Wald tests using different lag periods for $PM_{2.5}$ and O_3 and different O_3 metrics, presented by season

Pollutant	Health endpoint	Disease category	Temp cut-points in percentile (in °C)	p-value of the Wald test*		
				Benchmark (L02 and O_3 max-8h)	Replacing L02 with L01	Replacing O_3 max 8-h with O_3 max 1-h
Summer						
$PM_{2.5}$	Mortality	CVD	15, 70	0.40	0.26	NA
$PM_{2.5}$	Mortality	RD	(15.9,21.5)	0.60	0.42	NA
$PM_{2.5}$	ED visits	CVD		0.07	0.05	NA
$PM_{2.5}$	ED visits	RD		0.02	<0.01	NA
O_3	Mortality	CVD		0.18	0.18	0.13
O_3	Mortality	RD		0.58	0.61	0.63
O_3	ED visits	CVD		<0.01	<0.01	<0.01
O_3	ED visits	RD		<0.01	0.01	0.01
Winter						
$PM_{2.5}$	Mortality	CVD	15, 85	0.15	0.06	NA
$PM_{2.5}$	Mortality	RD	(8.4,11.9)	0.41	0.25	NA
$PM_{2.5}$	ED visits	CVD		0.06	0.11	NA
$PM_{2.5}$	ED visits	RD		0.01	0.04	NA

Note: * Testing the equality of coefficients at the different temperature strata (df=2)
CVD cardiovascular disease, *RD* respiratory disease

6.4 Discussion

To address Objective 2 of this thesis, a stratification model was fitted to explore whether there was effect modification by temperature of the effect of air pollution on acute health outcomes in the Melbourne Region. Based on this model, health risks of air pollution were quantified across the temperature range. The results of the analysis reported above suggested that temperature mainly modified the associations between exposure to $PM_{2.5}$ and O_3 and ED visits. In the Melbourne Region, there was strong evidence to support the existence of an interaction with temperature for the associations between $PM_{2.5}$ and respiratory ED visits, and between O_3 and cardiovascular and respiratory ED visits. By stratifying temperature into three strata for the summer, the risk estimates for respiratory ED visits related to $PM_{2.5}$ and for cardiovascular ED visits related to O_3 were highest in the upper temperature stratum. Surprisingly, the highest risk estimate for respiratory ED visits related to O_3 was found in the middle temperature stratum. In the winter, the association between $PM_{2.5}$ and respiratory ED visits changed significantly across the temperature range. Exposure to cold temperatures increased the risk for respiratory ED visits attributed to $PM_{2.5}$ as the risk estimate in the lower

temperature stratum was the highest and gradually decreased when the temperatures were warmer in the middle and upper temperature strata.

According to the results here, exposure to temperature extremes, both cold and hot, could potentially lead to more severe health effects associated with exposure to $PM_{2.5}$ and O_3. However, as can be seen from the risk estimates for respiratory ED visits associated with O_3 in the three temperature strata in the summer, the simultaneous exposure to temperature and air pollution may not necessarily result in a synergistic effect. This may be related to some other factors that were not considered in this analysis.

6.4.1 Plausible mechanisms and some evidence from laboratory studies

As there are shared patho-physiological responses and causal pathways for the health effects of exposure to air pollution and to temperature, it is possible that simultaneous exposure to these two environmental factors may have synergistic effects on health. It is well established that human thermoregulation involves sympathetic pathways of the autonomic nervous system (Witzmann 2009). Vasodilatation—one key response to increase heat loss from the body—is regulated by stimulation of sympathetic neurons embedded in the skin and blood vessels of the skin. Sweating—the other key mechanism to regulate body temperature—is controlled through postganglionic sympathetic nerves. Evidence from human studies on the health effects of $PM_{2.5}$ suggests that one pathogenic mechanism related to cardiovascular disease may be through increased tone of the sympathetic nervous system (Martinelli et al. 2013). This may lead to impaired cardiovascular function, resulting in, for example, arrhythmias and reduced heart rate variability. Hence, when the body is exposed to air pollution in an intense temperature environment, it is biologically plausible that a greater adverse health impact could be induced through these shared sympathetic nervous system pathways.

There have been few experimental studies designed to investigate responses to exposure to air pollution under a thermal stress. An animal study reported a synergistic response in rodents that were exposed to O_3 in a cold-stress environment (Watkinson et al. 2001). That study found that rats kept in an ambient temperature of 10°C with an O_3 concentration of 0.5 ppm had a decreased heart rate by a factor of two, compared to those at a room temperature of 22°C with the same O_3 concentration. In another laboratory study, the cell recovery process was prolonged in human malignant glioma cells exposed to a strong oxidising agent

when heat stress was also applied (Adachi et al. 2009). In this case, the oxidising agent was H_2O_2 but some air pollutants are also oxidising agents. Nevertheless, the relevance of these findings to normal cells in vivo is still unknown.

6.4.2 Comparison with other epidemiological studies overseas and Australian studies

Despite the limited evidence from laboratory and human studies, as reviewed in Chapter 2, there have been a growing number of epidemiological studies conducted in different world regions to investigate how temperature could alter the adverse health effects caused by air pollution exposure. These studies reported mixed findings, depending on the air pollutants studied and the setting of the study area.

The stronger associations between ED visits and exposure to $PM_{2.5}$ reported in this chapter along with decreased temperatures in the winter and increased temperatures in the summer are consistent with results from most previous studies conducted overseas. Four studies investigating how the association between PM_{10} and mortality varied across the temperature range in the US (Roberts 2004), Belgium (Nawrot et al. 2007), Italy (Stafoggia et al. 2008) and China (Qian et al. 2008; Li et al. 2011) reported enhanced PM_{10} effects on mortality at high temperature percentiles. For lower temperatures, a study in Scotland found strong evidence that temperatures lower than 11°C intensified respiratory mortality caused by exposure to black smoke (Carder et al. 2008). Similar findings were reported from a study in Shanghai, China (Cheng and Kan 2012), where there were stronger relationships between PM_{10} and all-cause, cardiovascular and respiratory mortality at temperatures less than the 15th percentile.

In part, the seasonal effect and the interdependence between PM and temperature may play a role and can be used to explain the stronger effect of exposure to PM at extreme temperatures. For example in winter when the temperature is low, PM levels can be high particularly in Melbourne due to the use of wood heaters and temperature inversions that trap air pollution near the ground as explained in Sections 3.4.4 and 3.4.5. As a result of the co-existence of these two variables, therefore there is the possibility of simultaneous exposure to extreme cold and high concentrations of PM which likely leads to a stronger health effect than exposure to either of them alone.

The increase in cardiovascular ED visits associated with exposure to O_3 when the temperature is elevated in the summer in the Melbourne Region, as found here, has also been observed in other foreign studies. A study in the UK suggested that a mortality risk associated with exposure to O_3 estimated in summers was stronger on hot days (Pattenden et al. 2010). Similar to the explanation given above for the high concentrations of PM in winter, the stronger health effects of O_3 found on hot days in summer may be due to co-existence of high levels of O_3 and hot weather. The co-existence of O_3 and high temperature in summer, as explained in Section 2.3, relates to the O_3 formation process that requires sunlight and warm temperatures. Among 15 conurbations considered in that study, the stronger effect of O_3 on mortality on hot days suggested that the effect of the interaction between O_3 and heat was significant only in London and Cardiff. The variation of the interaction between O_3 and temperature for the effect on all-cause and cardiovascular mortality across regions in the US was also reported in two studies analysing the NMMAPS data (Ren et al. 2009, 2008a). An interaction effect was apparent in the north-east, but not in the south-east. The difference in the interaction between these two regions was explained by actual personal O_3 exposure, which was influenced by the difference in climatic conditions, housing characteristics and the use of air conditioning (Chen et al. 2012).

In Australia, a number of studies have examined the modifying effect of temperature on the health effects of air pollution. Overall findings from these studies, including the results found here, were indicative of the existence of the modifying effect in some Australian cities. A study in Brisbane by Ren and Tong (2006) was among the first to address this issue in the country. Among six health outcomes considered in that study, the associations between five health outcomes and exposure to PM_{10} were stronger when temperatures were higher than the mean temperature. In Sydney, Hu et al. (2008) reported that the relationship between SO_2 and all-cause mortality in the summers during 1998 to 2004 was enhanced by exposure to high temperatures. In that study, when maximum temperatures were in the range of 29–32°C and SO_2 levels exceeded 3.15 ppb[3], the percentage increase in mortality was greater than when exposure was solely to temperatures above 32°C.

[3] That study originally reported the SO_2 level in the unit of pphm (part per hundred million) of 0.315. The level in pphm was converted here to the level in the ppb unit.

In Melbourne, two studies recently investigated the modifying effect of temperature on the association between all-cause mortality and exposure to PM_{10} and O_3 (Puza and Roberts 2013; Pearce 2011). The results from these two studies were contradictory. By applying a bivariate response-surface function together with a temperature-stratified model, Pearce (2011) found that the PM_{10} and O_3 effects associated with all-cause mortality in Melbourne were modified by temperature. For both pollutants, greater risks on mortality were apparent when temperature increased. In contrast, results from the study by Puza and Roberts (2013) did not indicate a significant modification effect of temperature on the association between PM_{10} and all-cause mortality in Melbourne. In that study, the authors applied a Bayesian approach that was believed to be superior to the traditional stratification approach in terms of taking into consideration statistical uncertainty related to the selection of temperature cut-points. Despite the absence of the interaction in Melbourne, there was significant temperature modification of the association between PM_{10} and all-cause mortality in Sydney, using the same Bayesian approach.

6.4.3 Explanations for unexpected results

There were inconsistent patterns of risk estimates across the temperature strata in the summer seen in the association between O_3 and cardiovascular and respiratory ED visits (see Figures 6.3c and 6.3d). The pattern for the O_3 effects associated with cardiovascular ED visits has been commonly found in other studies where a greater O_3 effect on health increases along with rising temperatures. However, a greater risk estimate for respiratory ED visits associated with O_3 in the middle temperature stratum rather than the upper temperature stratum has not been previously shown. This pattern could possibly be explained by indoor/outdoor (I/O) ratios[4] of air pollution in Melbourne, together with some evidence on how people respond to meteorological conditions.

Warmer temperatures result in increased time outdoors in summer in a temperate city like Melbourne (Dobbinson et al. 2008), up to around a maximum daily temperature of 28°C. On very warm days, there is a tendency to stay indoors, particularly for population groups whose thermal comfort range is relatively narrow. For example, a study in the elderly in Japan found

[4] The I/O ratio is a parameter used to represent the relationship between indoor and outdoor concentrations of a given air pollutant. A greater value of I/O ratio means a higher concentration of air pollution indoors than outdoors. This occurs when there is a source of air pollution indoors such as smoke from a cooking stove in a poorly ventilated house. A smaller value of I/O ratio may happen in a situation where the ventilation in the place of interest is very good and there is no indoor source of the air pollutant studied. A number of factors can influence the I/O ratio such as indoor sources, geometry of the cracks in buildings, outdoor wind environments, ventilation patterns and the use of filtration (Chen and Zhao 2011).

an increase in outdoor physical activity as temperature increased up to 17°C and then a decrease when the range of temperature was above 17–29°C (Togo et al. 2005). Thus it could be possible that at the upper temperature stratum (mean temperatures >21°C, based on the results in this chapter), Melbourne residents who had impaired health conditions or were sensitive to heat may prefer to stay indoors. As a result, the pollutants that they were exposed to would be mainly from indoor environments. According to a study in Melbourne, an I/O ratio of O_3 in summer/autumn (January to April) was approximately 0.13, which was considerably less than that for $PM_{2.5}$, which was 1.24 (The Centre for Australian Weather and Climate Research 2010). The low I/O ratio for O_3 is because this is a reactive agent that is removed by surface materials when it is transported from outdoors to indoors. In addition, there are no O_3 indoor sources. The O_3 concentrations people are exposed to when they stay indoors are much lower than when they spend time outside and are exposed to ambient O_3 concentrations. If the actual personal exposures to O_3 concentrations were lower in the upper temperature stratum, this could account for the lower risk estimate for respiratory ED visits associated with O_3, compared with the greater risk estimated in the middle stratum.

The pattern of risk estimates for cardiovascular ED visits was however different to that for respiratory ED visits, which is perhaps unexpected based on the explanation provided above. A recent integrated assessment for O_3 conducted by USEPA (2013, p 6-264) concluded that:

'the recent epidemiologic studies build upon and confirm the associations between short-term O_3 exposure and all-cause and cause-specific mortality reported in the 2006 O_3 AQCD [Air Quality Criteria Document]. *However, there is a lack of coherence across disciplines and consistency across health outcomes for O_3-induced cardiovascular morbidity which do not support the relatively strong epidemiologic evidence for O_3-related cardiovascular mortality.'*

One explanation for the findings shown here may be that O_3 is acting as a surrogate for PM pollutants, as discussed in Chapter 5, with the effects actually those of $PM_{2.5}$ on cardiovascular ED visits. In that case, even though people would prefer to stay indoors when temperatures are high (such as the temperatures in the upper temperature stratum of this analysis), they would still continue to be exposed to $PM_{2.5}$ because of the high I/O ratio discussed earlier. As a result, the risk estimated in the upper temperature stratum, which was

greater than the other temperature strata, could be the result of concurrent exposure to $PM_{2.5}$ under a heat stress condition.

6.4.4 Strengths and limitations of the analysis

The main strength of this analysis is the method used in selecting the temperature cut-points to define the temperature strata. Unlike other studies applying the stratification approach, in which temperature cut-points were mostly chosen arbitrarily, the temperature cut-points of the present analysis were selected from a large number of potential candidates in Step 1 of the analysis. Step 2 added another layer of accuracy to the selection process to ensure that the chosen cut-points were appropriate across multiple disease categories, health endpoints and pollutants. The robustness of the method can be seen from the results of the sensitivity analysis in that the temperature cut-points identified were mostly insensitive to the changes in model specification.

Other strengths of the present analysis include the long period of the time-series dataset as well as the use of the blending approach. The former provides greater statistical power to detect an association, if it exists, because it contains a higher level of day-to-day variation on both exposure and outcome variables accumulated over time. The use of the blending approach, as discussed earlier, has an advantage in reducing measurement error and consequently increasing the size of the risk estimate to be as close as possible to the true estimate.

There are a number of limitations in this analysis. The results have led to the conclusion that temperature modifies the effect of $PM_{2.5}$ and O_3 on ED visits, but not mortality. However, this conclusion has been drawn based on the data from a single city, and could be different if a multi-city approach was applied. Although there have been several studies designed to examine the interaction between temperature and air pollution in Australian cities, none of them have applied a multi-city approach. The study design of the multi-city approach, like the studies by Ren et al. (2008a, 2009) in the US, would help address the mixed results observed in the past Australian studies and could draw an overall conclusion on interaction effects between air pollution and temperature in Australia.

The strength of using the blending approach can become a limitation as well in terms of its limited capacity to predict ambient air pollution concentrations on days with extreme air

pollution events. As presented in Section 5.3, the $PM_{2.5}$ effect on mortality was detected when the data on bushfires and dust storms were included in the analysis and the standard approach was applied instead of the blending approach. It is possible that if the analysis relied on the standard approach and using all data, a significant interaction between temperature and $PM_{2.5}$ on mortality would have been observed. However, there have been on-going developments in modelling extreme air pollution episodes by TAPM-CTM (EPA Victoria and CSIRO 2013). Applying the blending approach in the future when the model is able to accurately predict air pollution concentrations during those events may provide better prediction of ambient exposures and subsequently health risk estimates associated with air pollution. This is particularly important in a country prone to air pollution events from natural origins like Australia.

Although the method used to test for a possible interaction between temperature and air pollution on health risks was quite robust, it is possible that the significant interactions are due to multiple testing and have occurred by chance. One way to overcome this issue is to use a Bayesian approach, as recently proposed by Puza and Roberts (2013), to handle uncertainties emerging from the process of selecting the temperature cut-points.

6.5 Applications of the relative risks estimates for future projections due to climate change

As stated in Chapter 1, the risk estimates derived from the analyses in this chapter will be used further in Component 2 of this thesis. To address Objective 3 in estimating changes in the health effects of air pollution due to changes in future air quality modified by climate change, the risk estimates from this point forward will be limited to those for respiratory ED visits because they were positive and statistically significant and there was no convincing evidence of increased risk of cardiorespiratory mortality or cardiovascular ED visits in the basic or stratified models. Since the projection of changes in future air quality undertaken in the Future Air Projections project for the current and two future time windows was limited to summers and winters, the analyses in Chapter 8 will also be specific to these two seasons. In line with the seasonal analysis, only the risk estimates for respiratory ED visits in the summer and in the winter reported in this chapter will be further used in Chapter 8. To explore how the use of risk estimates taking account of the effect modification by temperature would be different from those estimated from the conventional approach without inclusion of the

interaction, the risk estimates acquired from the basic and stratified models will be compared in Chapter 8.

In summary, three effect estimates from the basic and stratified models in Chapter 5 and this chapter respectively will be used to explore the change in future health risk estimations due to air pollution and climate change, namely, the effect estimates without and with temperature modification for:

- respiratory ED visits associated with $PM_{2.5}$ in the summer
- respiratory ED visits associated with O_3 in the summer
- respiratory ED visits associated with $PM_{2.5}$ in the winter.

As set out in Objective 4, these three risk estimates will also be used to examine how non-climate related factors and projection methods impact on the change in future health risks estimated in Objective 3.

6.6 Chapter summary

This chapter explored the potential of temperature as a modifier of the association between mortality and morbidity outcomes and exposure to air pollution in the Melbourne Region. This was achieved by fitting a stratification model. A number of temperature strata were defined and used to investigate variations in relative health risks related to $PM_{2.5}$ and O_3 exposure across different temperatures. The blending approach that was earlier identified as the best approach to estimate exposure was used in this chapter. When using an identified single pair of cut-points across disease categories, health endpoints and pollutants in each season, it was found that only estimated relative risks for ED visits, for both cardiovascular and respiratory, differed significantly between the temperature strata, suggesting the existence of a temperature modifying effect. The temperature modifying effect was not detected for the mortality outcomes. The pattern of estimated risks for ED visits found in the different temperature strata varied by health endpoint, type of pollutant and season. In the winter, by stratifying temperature into three strata, the association between respiratory ED visits and $PM_{2.5}$ was stronger in the lower temperature stratum. In contrast, the stronger association between respiratory ED visits and $PM_{2.5}$ exposure was found in the upper temperature stratum in the summer. For the O_3 effect in the summer, the risk for cardiovascular ED visits was strongest in the upper temperature stratum, whereas the risk for

respiratory ED visits was strongest in the middle temperature stratum. Results from a sensitivity analysis suggested that the identified temperature cut-points, the magnitude of relative risk estimates and the patterns of relative risks in the three temperature strata were insensitive to changes in the lag period of $PM_{2.5}$ and O_3 and the averaging time of O_3 when fitted in the stratification model.

The stronger health effects of air pollution at temperature extremes, both cold and hot, may be explained by stimulation of shared patho-physiological pathways, mainly the sympathetic nervous system, when the body is simultaneously exposed to these two environmental factors. The unexpected stronger association between respiratory ED visits and exposure to O_3 in the summer in the middle temperature stratum compared to the upper stratum may be explained by changes in behaviour to avoid exposure to extreme heat. In summer, when the temperature is high, people might spend more time indoors to avoid exposure to heat. As a result, they might be less exposed to O_3 as indoor O_3 concentrations are considerably lower than ambient concentrations, due to low permeability of ambient O_3 into indoor environments.

The risk estimates that differed in each temperature stratum derived from the analyses in this chapter will be used further in Chapter 8 to assess the health effects of air pollution due to climate change in the future. The further application of the relative risks derived from this chapter in Chapter 8 will be limited to the respiratory ED visits because the associations are clearest for this outcome. With regard to assessment of the impact of climate change on health in Chapter 8, there will be a comparison between estimated future changes in health risks related to air pollution based on the variable risk estimates across the temperature range with those using the uniform relative risks derived from Chapter 5.

COMPONENT 2

Assessing Impact of Climate Change on Future Health Effects of Air Pollution

Chapter 7 Literature review

This chapter serves as a review of literature for Component 2 of this thesis that aims at assessing the health risks of air pollution under the effects of future climate change. The focus of this chapter is to review approaches and methods used in past studies quantifying air pollution-related acute health outcomes under a changing climate. The approaches and methods reviewed will be used to guide the design of methods in Chapter 8 to address Objectives 3 and 4. In particular, gaps identified in the literature about uncertainties associated with projections of future health impacts of air pollution driven by climate change will be used to guide the uncertainty analysis for each stage of the health impact assessment conducted in Chapter 8 to address Objective 4.

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Quantifying the health impacts of air pollution under a changing climate—a review of approaches and methodology

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Abstract Climate change has been predicted to affect future air quality, with inevitable consequences for health. Quantifying the health effects of air pollution under a changing climate is crucial to provide evidence for actions to safeguard future populations. In this paper, we review published methods for quantifying health impacts to identify optimal approaches and ways in which existing challenges facing this line of research can be addressed. Most studies have employed a simplified methodology, while only a few have reported sensitivity analyses to assess sources of uncertainty. The limited investigations that do exist suggest that examining the health risk estimates should particularly take into account the uncertainty associated with future air pollution emissions scenarios, concentration-response functions, and future population growth and age structures. Knowledge gaps identified for future research include future health impacts from extreme air pollution events, interactions between temperature and air pollution effects on public health under a changing climate, and how population adaptation and behavioural changes in a warmer climate may modify exposure to air pollution and health consequences.

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Introduction

With increasing awareness of the threat posed to population health by climate change, there is demand from the public health community to quantitatively estimate the associated health burdens (World Health Organization 2009). Such quantification can help prioritise diseases and health outcomes, vulnerable groups, and affected populations so that appropriate policies and strategies can be developed. Rapid developments in climate science, particularly in the use of downscaling techniques, provide opportunities to investigate climate-related health consequences at the regional, state and city levels (Rosenthal et al. 2004).

The health impacts of air pollution are likely to be modified by climate change (World Health Organization 2003), due mainly to the exposure of populations to raised levels of air pollutants such as volatile organic compounds (VOCs), O_3 and some components of secondary particles. The emission and production rates of these substances can be enhanced in a warmer climate (Hogrefe et al. 2005). Future projections of surface O_3 and particulate matter (PM) have been undertaken more than other air pollutants because of their importance to public health (Ebi and McGregor 2008). Jacob and Winner (2009) summarized regional scale O_3 projections and found that future surface O_3 concentrations in the US and Europe could be increased by 1–10 ppb over the next century, particularly in polluted areas. On the other hand, future PM concentrations have been projected to vary with location. The authors attributed this variation to wide range of PM constituents and their different responses to changing meteorology. According to four studies conducted in US regions, precipitation was found to be an important determinate of future PM

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concentrations (Avise et al. 2009; Pye et al. 2009; Tagaris et al. 2007; Racherla and Adams 2006). Various sources of air pollution may respond differently to a future climate. For air pollution from natural sources, VOCs evaporated from vegetation may be increased with rising temperatures, as well as PM emitted from forest fires in areas projected to be drier (Flannigan et al. 2009). Similarly, changes in temperature can alter emissions from anthropogenic sources. For example, the reduced use of wood heaters in a temperate location can lead to a reduction of atmospheric PM concentrations for a warming climate trend (Cope et al. 2011b).

Despite the current importance of air pollution in determining disease burdens globally (Ostro 2004) and its sensitivity to climate change, only limited studies have attempted to quantify future health effects. This is partly due to the advanced and complex methods required in the quantification. Quantifying potential air pollution-related health effects requires air quality models (AQMs) to predict future air pollution levels based on climate model results, before they can be linked to health impacts functions. In addition, because of its interdisciplinary nature, such quantification demands collaboration from professionals in climate science, air quality and public health.

In this paper, we review methods that have been applied to quantify how future climate change will modify air pollution-related health effects. Past reviews focussed on study outcomes (Ebi and McGregor 2008; Kinney 2008; Barnett and Hansen 2009). In contrast, this review seeks to identify optimal approaches, and to suggest ways in which existing challenges can be addressed in order to attain improved health impact estimates.

Methods

Literature search and data extracted

We conducted a systematic search to identify published literature quantifying health impacts of air pollution and climate change. We searched five databases: PubMed, ProQuest Central, Science Direct, Scopus, and Web of Science. Search criteria were: (1) key words: climate change, global warming, future, air, O_3, particles, PM, mortality, and health; (2) studies published between 2000 and 2011; and (3) only peer-review journal articles, government reports, and conference proceedings. We also searched manually for relevant references in articles found. Based on the search criteria, 14 studies were included in this review (Table 1). One study each was published in 2001, 2004, 2006 and 2007, three studies in 2008 and seven studies during 2009–2011. The majority were peer-reviewed journal articles except two studies, which were a proceedings paper (Cope et al. 2011a) and a government report (Anderson et al. 2001). Although the focus of this review was on quantitative health impact estimations, we also included two studies (Anderson et al. 2001; Casimiro et al. 2006) in which future projections were not expressed as quantitative changes in health risk. Rather, the future trends were predicted descriptively based on mixed quantitative and qualitative analyses on future climate, air quality and health impacts. The approaches applied in these two studies are referred to as semi-quantitative in this review.

In each of the 14 studies, we examined its design, methods and, if available, results of sensitivity analyses to test associated uncertainties. Aspects of the study design we considered included study location, reference and projected time periods, and health effects (Table 1). Models and their corresponding scenarios applied to project climate, air quality and health impacts are listed in Table 2. A basic method commonly applied in all the reviewed papers is introduced in the following section on "General approach to quantifying health impacts associated with climate change" , followed by a section on "Approaches to quantifying the health impacts associated with air pollution and climate change", which briefly provides the setting, methods and scope of each study. A "Discussion" then compares the strengths and weaknesses of individual studies relative to the optimal approaches recommended for this research area.

General approach to quantifying health impacts associated with climate change

All papers reviewed applied a common basic method as outlined by Campbell-Lendrum and Woodruff (2007). This method involves two main elements: (1) using historical records to measure the effect of climate variation on health; and (2) applying known, or estimated, relationships to projected climate at a chosen future time. The first element involves developing a concentration–response function for each health outcome that is believed to be sensitive to weather and climate. This aim can be achieved via time-series studies. For the second, the International Panel on Climate change (IPCC) has developed a series of emission storylines and scenario families, referred to as Special Report on Emissions Scenarios (SRES). These scenarios are based on different population growth schemes, economic conditions, social development, progress in technology development and transfer among regions (IPCC 2000).

The two elements described above are sufficient if a relative percent change in the health outcome of interest is to be estimated. However, if a more complete picture of impacts on population health is the goal of the quantification, the information from the two elements needs to be

Table 1 Study locations, baseline and projected periods, and health effects quantified

Reference	Study location	Baseline (B) and projected (P) period	Health effect
Anderson et al. 2001	British Isles	B: 1990 P: decades in twenty-first century	O_3 and PM_{10}-related to unspecified health outcomes
Bell et al. 2007	50 US cities	B: 1993–1997 P: 2053–2057	O_3-related non-accidental, cardiovascular and respiratory mortality; hospital admissions for COPD; respiratory and asthma
Casimiro et al. 2006	Lisbon, Portugal	B: 1990s P: 2020s and 2050s	NO_2 and O_3 -related to unspecified health outcomes
Chang et al. 2010	19 US communities	B: 2000 P: 2041–2050	O_3-related premature mortality
Cheng et al. 2008	4 Canadian cities	B: 1981–2000 P: 2040–2059 and 2070–2080	Extreme temperatures, CO, O_3, NO_2, SO_2 and SP-related non-traumatic mortality
Cope et al. 2011a	Sydney region	B: 1996–2005 P: 2021–2030 and 2051–2060	O_3-related respiratory hospital admissions
Doherty et al. 2009	15 UK conurbations	B: 2000 P: 2020–2030	Heat and O_3-related premature mortality
Jackson et al. 2010	2 counties, Washington State	B: 1997–2006 P: 2045–2054	Heat and O_3-related non-traumatic and cardiopulmonary mortality
Jacobson 2008	World and the US	Comparing present days with preindustrial period	O_3-related mortality; hospitalisation and emergency-room visits; $PM_{2.5}$-related mortality; Non-methane VOCs-related cancer
Knowlton et al. 2008	New York region	B: 1990s P: 2020s, 2050s, and 2080s	Heat and O_3-related acute non-accidental mortality
Knowlton et al. 2004	31 New York counties	B: 1990s P: 2050s	O_3-related all internal causes mortality
Selin et al. 2009	16 world regions	B: 1999–2001 P: 2049–2051	O_3-related mortality; respiratory hospital admissions; respiratory symptom and minor restricted activity days; asthma; bronchodilator usage and lower respiratory symptoms
Sheffield et al. 2011	14 New York counties	B: 1990s P: 2020s	O_3-related childhood asthma
Tagaris et al. 2009	United States	B: 2001 P: 2050	O_3 and $PM_{2.5}$-related premature mortality; respiratory and cardiovascular hospital admissions; acute respiratory symptoms; respiratory emergency room visits; school loss days

CO Carbon monoxide, *COPD* chronic obstructive pulmonary disease, NO_2 nitrogen dioxide, O_3 ozone, $PM_{2.5}$ particulate matter with diameter 2.5 μm or less, PM_{10} particulate matter with diameter 10 μm or less, SO_2 sulphur dioxide, *SP* suspended particles, *VOCs* volatile organic compounds

linked further with the baseline mortality or morbidity rate and the population size. The standard formula generally applied in the studies we reviewed (Chang et al. 2010; Jackson et al. 2010; Jacobson 2008; Knowlton et al. 2004, 2008; Selin et al. 2009; Sheffield et al. 2011; Tagaris et al. 2009) is given below.

$$\Delta H = R * \left(e^{\beta * \Delta C} - 1\right) * Pop \quad (1)$$

where ΔH is the change in the health outcome of interest resulting from changes in an environmental factor, R is the baseline mortality or morbidity rate, ΔC is the estimated change in an environmental factor, β is the log relative risk associated with a change in exposure to the environmental factor, and Pop is the exposed population in the period and location of interest. From the point of view of future projections, different assumptions can be made about future rates of health endpoints R and populations Pop.

Approaches to quantifying the health impacts associated with air pollution and climate change

Studies attempting to estimate the health impacts, although sharing the same basis of the projections, have used different methods, each tailored to meet their goals and local context. Here we briefly summarise methods applied in individual studies that generally involve climate, air quality and health impact projections as depicted in Fig. 1. The output from a climate projection is coupled with an AQM to predict future air quality. The predicted change in air

Table 2 Climate, air quality and health models and their scenarios of the reviewed studies

Reference	Climate projection		Air quality projection		Health impact projection	
	Model[a]	Scenario	Model[a]	Scenario	Model	Scenario
Anderson et al. 2001	GCM - HadCM3	IS92a	Historical air pollution episodes	NA	Concentration-response function	NA
Bell et al. 2007	GCM - GISS RCM - MM5	SRES A2	CMAQ with SMOKE	Constant emissions	Concentration-response function	Population and age structure constant
Casimiro et al. 2006	GCM - HadCM2 RCM - PROMES and HadRM2	CO_2 concentrations two times larger than present	Historical air pollution episodes	NA	NA	NA
Chang et al. 2010	GCM - CGCM3 RCM - Canadian RCM	SRES A2	Statistical prediction models	Constant emissions	Concentration-response function	Population and age structure constant
Cheng et al. 2008	GCM - CGCMs, US GFDL- R30 with statistical downscaling and synoptic weather typing	IS92a, SRES A2 and B2	Within-weather-type historical simulation models	(1) Constant emissions; (2) decreasing 20 % and (3) increasing 20 %	Within-weather group mortality prediction models	Population and age structure constant
Cope et al. 2011a	GCM - CSIRO Mk3 RCM - CCAM	SRES A2	TAPM-CTM	(1) Constant emissions; and (2) decreasing 40 % and 70 % of O_3 precursors	Concentration-response function	Population and age structure constant. 60 ppb O_3 threshold
Doherty et al. 2009	11 coupled global chemistry-climate models and 15 global chemistry models simulated by numerical weather prediction data				Concentration-response function	NA
Jackson et al. 2010	GCM - PCM RCM - MM5	SRES A2	Global - MOZART-Regional - CMAQ with SMOKE	O_3 precursors based on the EPA Economic Growth Analysis System and land-use models	Concentration-response function	Projected population and age structure for 2025
Jacobson 2008	GATOR - GCMOM	(1) CO_2 at present-day conditions; and (2) CO_2 at the preindustrial period	SMVGEAR II	NA	Concentration-response function	35 ppb O_3 threshold
Knowlton et al. 2008	GCM - GISS RCM - MM5	SRES A2	CMAQ with SMOKE	Constant emissions	Joint concentration response function of temperature and O_3	Population and age structure constant
Knowlton et al. 2004	GCM - GISS RCM - MM5	SRES A2	CMAQ with SMOKE	(1) Constant emissions; and (2) growth in O_3 precursor emissions as identified in SRES A2	Concentration-response function	Population and age structure constant
Selin et al. 2009	GCM - GISS	SRES A1B	GEOS Chem	(1) Constant emissions; and (2) 2050 emissions	Concentration-response function	Population in 2050
Sheffield et al. 2011	GCM - GISS RCM - MM5	SRES A2	CMAQ with SMOKE	Constant emissions	Concentration-response function	Population and age structure constant
Tagaris et al. 2009	GCM - GISS RCM - MM5	SRES A1B	CMAQ with SMOKE	Constant emissions	Concentration-response function	Population and age structure constant

[a] Model names in full are available online (Annex 1)

CO_2 Carbon dioxide, *EPA* Environmental Protection Agency, *GCM* general circulation model, *O_3* ozone, *ppb* part per billion, *RCM* regional climate model

pollution is then used with a concentration-response function for predicting health impacts.

Two studies in this review focussed on estimations of the health impact associated with air pollution and climate change at the global scale. Jacobson (2008) compared mortality and morbidity due to exposure to O_3, $PM_{2.5}$ (PM with diameter 2.5 μm or less) and non-methane VOCs between present-day with preindustrial periods in which a near-surface temperature difference is 1.07 K. A general circulation model (GCM), GATOR-GCMOM, was nested with a box chemistry model, SMVGEAR II, to estimate differences of the pollutants between the two periods in this study. With the estimated changes of air pollutants, a concentration-response function for each pollutant derived from the literature was further applied to quantify the health impacts assuming a 35 ppb O_3 threshold and zero thresholds for the other pollutants. The other study (Selin et al. 2009) examined relative changes in health impacts and the associated costs due to potential increased O_3 for the period 2049–2051 in 16 world regions. Greenhouse gas (GHG) emissions based on the IPCC SRES A1B scenario were used to simulate a GCM, GISS GCM3. Projected climate was input to a global atmospheric and chemical transport model, GEOS-Chem, for estimating baseline and future O_3 concentrations. Emissions of O_3 precursors were held fixed at the stage of O_3 projection. The health impact was quantified by using concentration-response functions gathered from a collection of original time-series and meta-analysis studies.

This research area has been more active in North America than the other regions. Knowlton et al. (2004) pioneered estimating future mortality due to O_3 under a changing climate at the regional level in Metropolitan New York. To achieve the regional projection of climate and O_3, a GCM, GISS, forced by IPCC SRES A2 was linked with a regional climate model (RCM), MM5, prior to integrating future simulated climate to a chemical transport model, CMAQ. The projections for the 2050s were compared with the reference period, 1990s. The projected O_3 concentrations were applied to assess relative changes in the mortality based on a concentration-response function that was pooled from seven epidemiological studies with an assumption of a constant population and mortality rate over time. The same

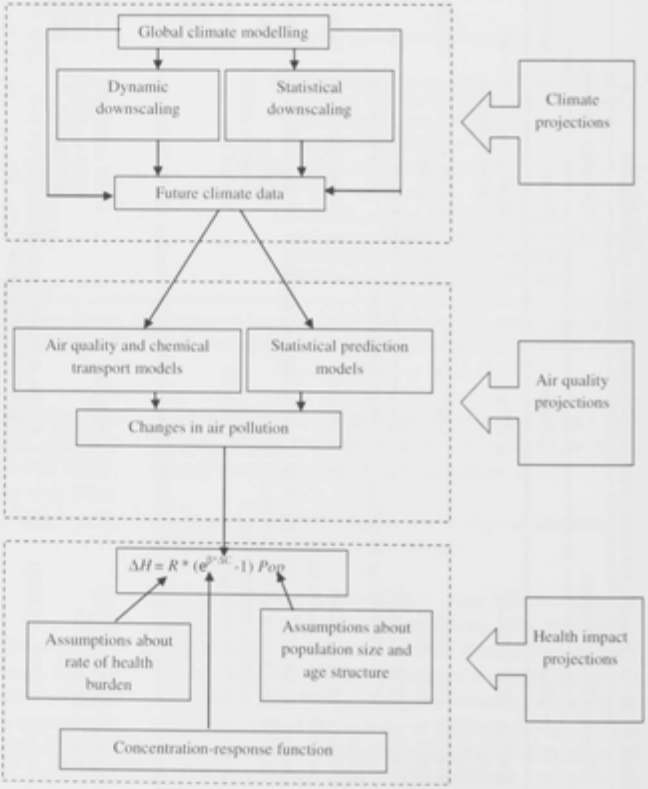

Fig. 1 Major steps for projecting health impacts associated with air pollution and climate change. ΔH Change in health outcome resulting from changes in air pollution exposure, R baseline annual mortality or morbidity rate, β log relative risk associated with a unit change in air pollution exposure, ΔC estimated change in air pollution concentration, Pop future exposed population

projection method was also employed in two other studies assessing impacts of joint exposure to heat and O_3 on mortality risk (Knowlton et al. 2008) and O_3-related childhood asthma (Sheffield et al. 2011) in the same location. This method was again applied in the study of Bell et al. (2007) to quantify relative changes in O_3 concentrations and related mortality and hospital admissions between the reference period, 1993–1997, and a future period, 2053–2057, in 50 US eastern cities. Tagaris et al. (2009) modified this method slightly by using IPCC SRES A1B to drive the GISS global model and expanding the projection to assess future $PM_{2.5}$ concentrations and associated health effects across the United States. Similarly, a slightly modified framework of climate and O_3 modelling system from the studies above was used by Jackson et al. (2010), who downscaled a GCM, PCM, and a global chemistry model, MOZART-2, to the MM5 regional climate model and the regional chemistry model CMAQ for projecting O_3 related-mortality at mid-century in two counties of Washington State. The O_3 projection was based on an assumption of changes in O_3 precursors according to future economic growth. In that study, future population growth was factored in with the use of population estimates for the years 2005–2030.

Chang et al. (2010) and Cheng et al. (2008) employed different methods from the other studies in the North American region when predicting health impacts from future changes in air pollution and climate. Chang et al. (2010) projected O_3 levels in southeastern US in the 2040s by using the regional climate output from a coupled Canadian global-regional climate modelling system driven by IPCC SRES A2 scenario to fit a statistical prediction model. Thereafter a concentration-response function was obtained from a multi-city study to project mortality attributed to the future change in O_3 assuming population and age structure held fixed at the baseline period (2000). Cheng et al. (2008) applied a synoptic weather-typing approach to predict impacts of future extreme temperatures and air pollution on mortality in two time windows (2040–2059 and 2070–2089) in four cities in south-central Canada. To predict climate and air pollution, output from an ensemble of multiple GCMs driven by three IPCC scenarios was downscaled statistically before being linked with a statistical prediction function corresponding to each meteorological and air pollution related-weather type. The prediction functions were developed through grouping daily historical observations that caused high air pollution levels and temperatures. Predicted air pollution and weather of each weather type were then used as predictors in a within-weather-group mortality prediction model. This study projected the impacts based on three future scenarios of air pollution emissions including the baseline during (1981–2000), 20 % higher and lower of the emissions than the baseline.

Regional projections of climate change impacts on health associated with air pollution have also been undertaken in some European countries and in Australia. Doherty et al. (2009) simulated O_3 concentrations and temperatures across the UK by using an ensemble of coupled global climate-chemistry models for the baseline period (1993–2003) with likely three pollution emissions scenarios. The projected information was combined with risk estimates for these two environmental factors in 15 conurbations evaluated during the baseline period to project future mortality for 2030. In Australia, Cope et al. (2011a) projected relative changes in hospital admissions attributable to O_3 exposure in Sydney Region between 1996 and 2005 and two future periods, 2021–2030 and 2051–2060. A chemical transport model, TAPM-CTM, was linked with a coupled global-regional climate modelling system forced by IPCC SRES A2 to predict meteorological and pollution fields at a 3-km grid spacing over the area of interest. When projecting the health effects, this study assumed constant anthropogenic O_3 precursors emissions and population over time.

Projections made by Anderson et al. (2001) and Casimiro et al. (2006) can be described as semi-quantitative approaches. Anderson et al. (2001) projected the future occurrence of summer and winter air pollution episodes out to 2100 on the basis of changes in weather variables under an IPCC business-as-usual scenario, IS92a, along with possible trends in pollutant emissions. The predicted future occurrence of air pollution episodes for the British Isles was considered together with a baseline health estimate of PM_{10} (PM with diameter 10 μm or less) and surface O_3 to describe future trends of the health impacts. Casimiro et al. (2006) examined recorded meteorological conditions during the 1990s and associated them with pollutant episodes for NO_2 and surface O_3 in Lisbon, Portugal. Climate output from two RCMs driven by an anticipated future CO_2 emissions scenario for two time periods, 2020s and 2050s, was used and assessed possible NO_2 and O_3 air pollution episodes. Despite a lack of baseline data on health burdens associated with air pollution, the study was able to qualitatively estimate potential changes in health outcomes attributable to NO_2 and surface O_3.

Results and discussion

In this section, we analyse and discuss strengths and weaknesses of the methods described above for each stage of the projections undertaken in the 14 studies. We also identify how these studies handled uncertainty, which is a significant issue when projecting future impacts of climate change.

Climate projections

At this stage of the projection, a number of procedures have been recommended to cope with model and GHGs emissions uncertainties. It is widely recognised in climate science that one approach to address climate model uncertainty is to use an ensemble of models (Bader et al. 2008; Meehl et al. 2007). However, this strategy brings in a requirement of massive computational resources. Practically, therefore, among the studies we examined, only two studies were able to implement the model ensemble strategy (Doherty et al. 2009; Cheng et al. 2008). The use of global scale projections only, without exploiting additional resources for generating fine-resolution climate information, was the key factor that made it possible for Doherty et al. (2009) to use an ensemble of 26 global atmospheric chemistry models. The remaining studies were based on the use of a single GCM following by (for most studies) a dynamic downscaling approach, which is also computationally expensive. In the reviewed studies, it is likely that the process for selecting a single GCM was based primarily on the capacity of an individual GCM to best reproduce the climatology in the area of interest. For example, Bell et al. (2007), Knowlton et al. (2008) and Tagaris et al. (2009) used GISS—a GCM developed by the US Goddard Institute for Space Studies—in their research to project the health impacts of American populations. Likewise HadCM3—a GCM developed by a UK institution—was used to project health impacts in Britain and Portugal (Anderson et al. 2001; Casimiro et al. 2006). The projections made using these single GCMs, and the associated uncertainty will be best quantified for the study regions, where the output from the GCMs used was verified against historical observations. In any event, the use of projections from a single GCM has the potential to introduce bias, due to not accounting for uncertainty related to climate model physics. In an assessment of heat-related mortality from climate change in six cities, model bias was found to be larger than the uncertainties associated with future GHGs scenarios and downscaling (Gosling et al. 2012). Therefore it is strongly recommended that ensembles of models—'multi-model ensembles' or 'perturbed physics ensembles'—should be used for projection studies where practical (IPCC 2007). Although Cheng et al. (2008) considered a regional impact, the authors were able to include projections from three GCMs. This is because this study applied regression-based statistical methods, which are more computationally efficient compared to the dynamic approach, in downscaling the GCM.

GCM climate change simulations are forced by a GHG emissions scenario. Again, using multiple future GHG emissions scenarios is the favoured approach to address GHG emissions uncertainty, particularly when making projections close to the end of the century where the emissions have a greater degree of uncertainty (Carter et al. 2007). However, this approach is certainly offset by the computational resources required to run the same model multiple times. Based on the studies we reviewed, only Cheng et al. (2008) and Knowlton et al. (2008) used the output of GCMs forced by more than one IPCC SRES scenario. These two studies purposely selected the IPCC SRES B2 as an alternative scenario representing a lower bound of GHG emissions in comparison with other IPCC SRES scenarios representing high GHG emissions. Within a medium timeframe, surprisingly, Knowlton et al. (2008) found a change in 2050s relative to 1990s for O_3-related mortality estimated for New York City Metropolitan Region by using the B2 scenario, low emissions, 3 % larger than the A2 scenario, high emissions. Cheng et al. (2008), although stated making projections over two future time periods—2050s and 2080s—with the use of three different IPCC scenarios—IS92a, A2 and B2—did not provide detailed estimates of air pollution-related mortality for each scenario corresponding to each time period. In the same study, however, the authors found a small discrepancy of approximately 3 % for the period 2050s while comparing an average of percentage change across four Canadian cities for heat-related mortality driven by the A2 with B2 scenarios. The discrepancy became significantly greater (55 %) for the projected period 2080s. The results from these two studies suggest that only a small discrepancy of the health impacts between high and low GHG emissions scenarios can be expected if the time horizon for the projections is not beyond 2050s. This also suggests that relying on only a single GHG emissions scenario for studies aiming to project the health impacts when the focus of their projected time periods is before 2050s, therefore, would not significantly impact the projection results.

Air quality projections

Following the generation of climate projections, meteorological fields are linked with an AQM. Similar to climate modelling, running multiple AQMs would ideally help detect uncertainties associated with diverse simulation processes and functions handled by different modelling systems. Likewise, projecting future air pollution under multiple likely pollution emissions scenarios would be a desirable way to handle uncertainties associated with future emissions.

As can be seen from Fig. 1, air quality projections can be achieved through either numerical or empirical modelling. In our review, most studies employed three-dimensional numerical chemical transport models (Bell et al. 2007; Cope et al. 2011a; Doherty et al. 2009; Jackson et al. 2010; Jacobson 2008; Knowlton et al. 2004, 2008; Selin et al. 2009; Sheffield et al. 2011; Tagaris et al. 2009). The ability

of the numerical models to handle complex flows and non-linear chemical reactions of air pollution likely makes this approach more favourable. However, these advantages are offset by the high computational demands of such models. Consequently, this appears to limit the development of an AQM ensemble and thus becomes a constraint on exploring the uncertainty associated with AQMs. At the global scale, three of the reviewed studies employed single chemistry-climate models to simulate effects of the change in GHG emissions on global air quality (Jackson et al. 2010; Selin et al. 2009; Jacobson 2008). Only one study was able to simulate results of global surface O_3 projections from an ensemble of atmospheric chemistry-climate models (Doherty et al. 2009). At the regional scale, all the studies projecting regional air quality were dependent on a single chemical transport model (Bell et al. 2007; Knowlton et al. 2004, 2008; Jackson et al. 2010; Sheffield et al. 2011; Cope et al. 2011a).

Application of a statistical prediction model for projecting future air quality was found in two studies (Cheng et al. 2008; Chang et al. 2010). Chang et al. (2010) developed a linear regression model relating three meteorological variables, namely total cloud cover, solar radiation, temperature, as predictors to the prediction of surface O_3 concentrations during the year 2000 to forecast future surface O_3. Although fundamentally Cheng et al. (2008) also developed statistical prediction models for predicting multiple air pollutants under global climate change, the approach in their study was different and more complex compared to the study of Chang et al. (2010). Due to the advantage of inexpensive computer resources of these empirical modelling schemes, similar to the statistical downscaling strategy, studies adopting them would have more opportunities to conduct analyses on uncertainty from various factors, including model uncertainty, contributing to changes in future air quality (Chang et al. 2010).

With regard to future pollution emissions scenarios, we found that the most common approach was to assume constant anthropogenic emissions. Less common, but applied in a few studies, for example the studies of Knowlton et al. (2004), Sheffield et al. (2011) and Selin et al. (2009), involved setting up emission projections consistent with the storylines identified in the IPCC SRES scenarios. Apart from these two approaches, some studies built an emissions scenario on the basis of trends in current technologies and other factors contributing to the emissions. For instance, Doherty et al. (2009) simulated surface O_3 predictions based on three possible futures, one of which was a low emissions scenario assuming the implementation of currently available emissions control technologies (Dentener et al. 2005). Jackson et al. (2010) developed an emissions database to predict future air quality based on projections of economic growth and changes in land use for the period 2045–2054. Cope et al. (2011a) set up two future pollution emissions scenarios—assuming 40 % and 70 % reductions in O_3 precursors emissions relative to the reference period—to evaluate the achievement of compliance with the current standards for O_3 in the 2050s.

The literature we explored presented a variety of findings when air pollution emission projections were factored in to future air quality. Knowlton et al. (2004) and Sheffield et al.(2011) found a reduction in future O_3 concentrations and health impacts alike when an increase in anthropogenic O_3 precursor emissions was considered in conjunction with the impact of climate change. The authors explained that the reduction in O_3 concentrations was likely to be associated with titration of O_3 by higher concentrations of NO_x. On the contrary, assuming the growth of anthropogenic O_3 precursor emissions in the study of Doherty et al. (2009), projecting 2030 O_3 for the entire UK, resulted in an additional O_3 increase of 14 % relative to when the scenario of climate change alone with holding the emissions constant at the present level was considered. Likewise, Selin et al. (2009) estimated an average O_3 increase across regions globally for 2050s of approximately 6 ppb under the assumption of increased O_3 precursor emissions and a changing climate in addition to the isolated climate change impact. The diversity of findings from these studies may be due partly to different spatial resolutions in the projections and the areas projected, which led to differences in the magnitude of air pollution concentrations and corresponding density of populations exposed. However, they strongly demonstrated a large contribution of uncertainty associated with future air pollution emissions in air quality projections and a need to examine it in the projection process.

Health impact projections

In quantifying the impacts of climate change on health, the critical steps that must be undertaken with care involve choosing concentration–response functions and considering likely future population scenarios. If chosen from the literature, concentration–response functions for major health outcomes should be from combining multiple well-designed epidemiological studies. It is also important to demonstrate uncertainties associated with a chosen concentration–response function through a sensitivity analysis as the chosen function will be certainly subject to change in the future, which may impact greatly on the projected results. To isolate the magnitude of impacts of climate change and future population demographics on air pollution-related health, varying these factors can be undertaken as part of sensitivity analyses.

Most studies we examined used a concentration–response function that was estimated based on a multi-city study or meta-analysis. Between these two approaches,

using health effect estimates from multi-city studies is recommended due to the unavoidable publication biases associated with meta-analyses (Bell et al. 2005). Rather than using a single estimate, Bell et al. (2007) and Jacobson (2008) chose a set of estimates from a number of epidemiological studies for a given health endpoint. According to Bell et al. (2007), different concentration–response functions for a given health outcome could lead to a two-fold difference in percentage changes in health outcome induced by exposure to O_3 and climate change for 2050s relative to the baseline period 1990s. Similarly Selin et al. (2009), with the application of Monte Carlo analysis to measure sensitivity to the concentration–response function for O_3-related mortality, found that the limits of a 95 % probability interval differed by a factor of two. These findings indicate the importance of the choice of concentration-response function. This same issue was highlighted in a study of climate change and diarrheal disease that found that the choice of concentration–response function was more important than the choice of climate model (Kolstad and Johansson 2011).

Despite strong evidence of the health effects of long-term exposure to air pollution, only one study conducted by Jacobson (2008) explored the effect of increased exposure to non-methane VOCs on cancer in relation to climate change. This is an area that future studies should take into account to avoid misleading underestimations of the health impacts.

Alternatives to taking a concentration–response function from the literature are to estimate the function based on local data (Knowlton et al. 2008; Chang et al. 2010; Cheng et al. 2008). Despite using local data, Chang et al. (2010) applied the principle of multi-city study to identify a relationship of surface O_3 and mortality through combining a relative risk estimate for each of the 17 US counties examined. Cheng et al. (2008) used a synoptic weather typing approach, to identify a within-weather-type health prediction function based on local data. Knowlton et al. (2008) had to rely on local data in estimating a joint relationship of O_3 and temperature on mortality because such relationship had rarely been explored previously.

The frequency of extreme events involving simultaneous exposure of a population to high temperature and air pollution has been increasing since late twentieth century (Dear et al. 2005; Tong et al. 2010; Filleul et al. 2006). Such events are projected to be more frequent and intense in a warmer future climate (Clark et al. 2006). However, based on our review, this concern has gained modest attention. Among the existing studies, only one study attempted to estimate health impacts of interactions between temperature and air pollution in a future population (Knowlton et al. 2008). Other studies, although quantifying both the future health effects of heat and air pollution due to climate change, did not consider a combined effect of these two environmental risk factors (Doherty et al. 2009; Cheng et al. 2008; Jackson et al. 2010).

Other factors can also cause uncertainties at the stage of health impact projections. Some of these are associated with incomplete knowledge in estimating the concentration-response functions (Ren and Tong 2008). One example is the adverse health effects due to O_3 exposure at low concentrations. Many studies have found linear relationships between exposure to criteria air pollutants and adverse health effects, with no threshold. However, a few have argued that PM and surface O_3 exhibit non-linear exposure-response curves but with thresholds lower than current standards (Bell et al. 2006; Stylianou and Nicolich 2009). Different approaches to applying an O_3 threshold in the health impact function found in our review reflect these on-going debates. Predominantly, the studies examined in this paper applied a linear concentration–response function of O_3 exposure with zero thresholds (Anderson et al. 2001; Bell et al. 2007; Chang et al. 2010; Jackson et al. 2010; Knowlton et al. 2008; Selin et al. 2009; Sheffield et al. 2011). Conversely, the studies of Cope et al. (2011a) and Jacobson (2008) used non-zero thresholds. To deal with the uncertainty of the O_3 threshold on health impact predictions, a sensitivity analysis could be conducted to determine how applying a threshold level would affect final health estimates. For example, Knowlton et al. (2004) included a 20-ppb O_3 threshold in one of their sensitivity analyses. With the threshold assumption, a slight larger climate-driven increase in O_3-related mortality was found.

Estimating the future health burden in a particular location requires making assumptions about population demographics, and baseline mortality or morbidity rates of interest. While most studies we examined held these factors constant over the projected time period, a few studies chose to simultaneously bring population growth to play in the quantification, so that a joint effect of future changes in population size and the impacts of climate change was determined explicitly (Jackson et al. 2010; Selin et al. 2009). This is important as the uncertainty originating from future population growth has been identified to have the greatest influence, among other uncertainties associated with health impact projections, on the results of projecting the health impacts (Knowlton et al. 2004; Sheffield et al. 2011). As clearly shown from a sensitivity analysis conducted by Knowlton et al. (2004), which took into account population growth corresponding to the IPCC SRES A2 scenario, an estimate of O_3-related mortality for the period 2050 was increased by more than 50 % relative to considering just the impacts of climate change or the combined impacts of climate change and O_3 precursor emissions. Similarly, although to a lesser extent, Sheffield et al. (2011) found an additional 3.3 % of future O_3-related respiratory emergency department visits in children for 2020s

when an age-specific future population projection was taken into account together with the effect of climate change. Although it is well known that the health risks are higher among the elderly and children, this study was the only one, among the others we reviewed, that applied an age-specific concentration-response function and included information not only on the growing population size but also on future age demographics. Considering changes in size and age demographics are both equally imperative as many countries, even some developing countries, are transitioning to an aging society.

Conclusions

Estimating the health impacts of air pollution and climate change involves linking climate, air quality and health projections. This interdisciplinary area of research is still at an early stage in its development as can be seen from the limited number (only 14) of studies found from the literature search. Additionally, O_3 has been the primary focus of this research, with only a limited amount of work done on other pollutants such as PM. Although facing technical challenges, recent studies have developed methods that are applicable to different conditions and can improve reliability and transparency of the prediction results. The following is a summary of approaches commonly applied and a recommendation of methods for providing credible health estimates based on pros and cons of the studies we examined. Areas that will help improve the health impact estimation, but have received minimal or no attention yet, are identified at the end of this section.

In the studies we reviewed, climate projections were based mostly on a single GCM driven by a single IPCC GHG emissions scenario such as A1B or A2. When a regional projection was undertaken, a single RCM was used for dynamic downscaling. Clearly, a high demand for computational resources in running these numerical models results in reduced opportunities to investigate the variability of climate models and future GHG emissions, particularly when output at higher resolution for projecting regional climate is required. To overcome this challenge, the statistical downscaling technique, particularly when used in combination with statistical prediction models in the later stage of projecting air pollution, offers an alternative approach with computational efficiency. This approach allows flexibility in running a GCM model ensemble and comparing multiple future GHG emissions scenarios, a critical step in making projections close to the end of the century, while exploring other future uncertainties. In relation to the choice of future GHG emissions scenarios, we recommend the use of the IPCC SRES business-as-usual scenario such as A2 or A1B if the time horizon for the projections is not beyond 2050s. If the coverage of a projected time span goes beyond 2050s, at least two scenarios representing low and high emissions should be compared. However, these recommendations on the choice of IPCC scenarios might be subject to change due to development of four new key scenarios of future GHG emissions that will replace the SRES (Moss et al. 2010).

For air quality projections, the common approach was to integrate the projected climate information into air quality numerical models while holding future air pollution emissions constant. Taking advantage of advances in downscaling techniques, we found an increasing trend towards the use of regional air quality projections. This effort should be continued as it is critical to long-term air quality management. Taking into account different air pollution emissions scenarios should, from our perspective, be part of the air quality projections. This is particularly important in highly polluted areas with rapidly growing trends in economic and industrial development.

The most common approach in the health impact projections involved taking a concentration-response function from published literature and assuming no change in the current population and background health outcome rates. With respect to the choice of concentration–response function, we recommend the use of a relative risk function derived from a multi-site study. We also recommend conducting a sensitivity analysis to explore uncertainty from different concentration–response functions. In terms of population scenarios, we recommend consideration of changes in future demographics, both size and age structures. In case the full exercise of quantifying the impacts of climate change on air quality and health at local scale cannot be undertaken, at the very least, a semi-quantitative approach is recommended.

Three topics with regard to quantifying the health impacts should be priorities for future research. The first priority is about estimating future health impacts of extreme air pollution events including forest fires and dust storms. It is clear that there are strong associations of major forest fires and dust storms with a range of health outcomes (Analitis et al. 2012; Hashizume et al. 2010). There is also a possibility that climate change may cause increases in the occurrence of these extreme air pollutions as the century progresses (Flannigan et al. 2009; Aldersley et al. 2011). Although still premature in the current research, some progress has been made in projecting future air pollution extreme events in particular forest fires (IPCC 2007; Carvalho et al. 2011). Therefore, future studies should consider incorporating the health impacts of extreme air pollution events in the quantification. The second priority concerns behavioural adaptation of populations to cope with a warmer climate, which may modify exposure to air pollution. In projecting future

temperature-related health effects, these changes have been important factors commonly taken into consideration (Kinney et al. 2008; Gosling et al. 2009). Some of them, such as opening windows and spending more time outdoors, have potential to alter not only the health risks of heat but also health impacts of air pollution (Barnett and Hansen 2009). The last priority is to investigate interactions between temperature and air pollution. Although still limited to date, we did find one study in this review that attempted to factor in the combined effects in the projection. Further advances in this research area should take advantage of a growing body of empirical relationships derived from epidemiological studies of the interactive effects (Qian et al. 2008; Ren et al. 2011).

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Chapter 8 Analysing the impact of climate change on future health effects of air pollution

8.1 Introduction

One of the potential impacts from climate change that has been identified in a number of assessments is degradation of ambient air quality, with consequent health risks (Portier CJ et al. 2010; World Health Organization 2003; European Environment Agency 2005; Confalonieri et al. 2007). In order to plan actions to protect the population's health from the risks of deteriorating air quality as a result of climate change, evidence is needed on the likely magnitude of the impacts. The goal of this chapter, therefore, is to quantitatively estimate the impact of climate change on acute health risks attributable to changes in air quality in the Melbourne Region.

This chapter is organised and presented in two main parts to address Objectives 3 and 4 of this thesis respectively. In the first part, the estimated health risks presented in Chapters 5 and 6 are combined with the predictions of future air quality driven by climate change made by the Future Air Projections project. The scope of this first part is limited to estimations of acute health impacts attributable to air pollution modulated by climate change alone. As demonstrated in Chapter 6, the health risks associated with air pollution can vary across the range of ambient temperature. Thus estimations of air pollution-related health impacts from climate change that also take account of effect modification by temperature are also reported in the first part of this chapter. Methods and results of this first part are presented in Section 8.4 while the corresponding discussion is included in the first part of the discussion section, Section 8.6.1.

Uncertainty is intrinsic to predicted impacts from climate change. As discussed in the previous chapter, analysing the sources of uncertainty and their effects on the estimates also contributes to the evidence base. The second part of this chapter, thus, presents an assessment of uncertainties which may arise during the estimations made in the first part of the chapter. A number of factors and projection methods which can cause uncertainty in quantifying health risks associated with air pollution and climate change are explored and discussed. Again this part of the chapter builds on the work undertaken by the Future Air Projections project which examined a range of scenarios for air pollution emissions and demographic changes in the study area. Methods and results of this second part are presented in Section 8.5 while the corresponding discussion is included in the second part of the discussion section, Section 8.6.2.

Since this chapter builds upon the Future Air Projections project, the background of the project is briefly presented in the following section. This section also outlines how, and the extent to which, the project's outputs and results are employed and linked to address the relevant objectives of this thesis. The data to be used in the analyses of this chapter are described in Section 8.3.

8.2 The Future Air Projections project

8.2.1 General background

The Future Air Projections project was an initiative of the EPA Victoria. The project was undertaken in partnership with the CSIRO. The aim of the project was to explore the impacts of climate change, emission trends and population trends on future air quality under a range of scenarios in the state of Victoria, Australia. The project involved four key components: i) projecting changes in future air quality modulated by climate change alone in the PPR (Melbourne and Geelong) for two time windows centred around 2030 and 2070; ii) identifying other factors which could influence future air quality in the 2030s in the PPR and predicting air pollution concentrations and population exposure for that time period; iii) assessing the effect of climate change on the occurrence of windblown dust events and consequent air quality in Victoria for the period 2065 to 2074; and iv) developing and verifying methods for modelling the effects on air quality of bushfires in Victoria and assessing the plausibility of predicting how bushfires will affect air quality in the future.

The outputs and results of the first two components were employed in the estimations of the health impacts associated with air pollution under climate change scenarios assumed in this chapter. The following two sections (Sections 8.2.2 and 8.2.3) briefly outline the methods applied and some key results of the two project components which are relevant to the content of this chapter. Full details of the project are described in a series of publications and a project report (Walsh et al. 2013; Cope et al. 2011b; Cope et al. 2011c; EPA Victoria and CSIRO 2013).

8.2.2 Projections of air quality as a result of climate change alone, in the Port Phillip Region

To predict air quality under a changing climate in the PPR for the two future decadal periods—2025 to 2034 (decade 2, the 2030s) and 2065 to 2074 (decade 3, the 2070s)—the project selected the A2 SRES GHG emissions scenario to force four GCMs: CSIRO, ECHAM5, GFDL and UKMO. A prediction for the period 1996 to 2005 (decade 1, the 2000s) was also made as a baseline. Meteorological results from the four GCMs were dynamically downscaled by a RCM—CCAM—to a grid resolution of approximately 60 km which covered the Australian region. These global-regional model results were subsequently downscaled to a grid resolution of 3 km over the PPR using a chemical transport model (TAPM-CTM). The meteorological conditions generated from these coupled models were then used to simulate air quality, using an emissions inventory for 2006. As noted in Chapter 1, this project component considered solely the impact of climate change on air quality (Climate Penalty); therefore, it was assumed that there would be no changes in air pollution emissions or population between the decades. Note however that the dynamic effect of temperature on emissions from vegetation and motor vehicles was included in this part of the study, reflecting an important aspect of how climate change affects air quality. Simulations for two summer months (January and February) and two winter months (July and August) were made using the four GCMs in each year for decades 1 and 3. Simulations were generated for every month in all three decades using the GFDL modelling system. The air pollutants of interest in this project component included CO, NO_2, O_3, $PM_{2.5}$ and SO_2.

The results from this project component suggested that O_3, among the air pollutants considered, was the most sensitive to changes in climate. According to the summer month simulations, an increase in population exposure to O_3 max 1-h concentrations between decades 1 and 3 was predicted by all four GCMs within a range of 15% to 30% (EPA Victoria and CSIRO 2013). Note that predictions of population exposure to O_3 max 1-h concentrations in this part of the project were based on the assumption of a health-effect threshold level of 25 ppb; exposure below this level was ignored. The $PM_{2.5}$ 24-h avg concentrations in summer between decades 1 and 3 were also predicted to rise under all four GCMs, ranging from 4% to 13% in population exposure. In the winter simulations, the GFDL model was the only one that predicted a 10% increase in $PM_{2.5}$ population exposure. In contrast, a 2% to 5% decrease in $PM_{2.5}$ population exposure was predicted by

the other three GCMs. The underlying drivers for the air quality changes in the summer were largely related to a rise in temperature at approximately 2°C and a decrease in ventilation rate. Among the four GCMs, a highest reduction in ventilation rate and a smallest increase in temperature was predicted by the GFDL model for the winter simulations, explaining the exception of the increased $PM_{2.5}$ in the winter (Cope et al. 2011c).

8.2.3 Air quality projections based on a combination of changes in climate, air pollution emissions and population in the Port Phillip Region

The second component of the Future Air Projections project explored future air quality under different scenarios projected for the period 2025 to 2034. Other than the pollutants included in the first project component, a group of hazardous air pollutants (benzene, formaldehyde, toluene and xylenes) was also studied.

In this project component, changes in two non-climate factors—anthropogenic emissions of air pollutants and population growth—were identified as potentially affecting future air quality. In line with potential changes in emissions of air pollutants and population growth, three plausible emissions scenarios and corresponding inventories were developed and referred to by the project as 'low impact future', 'medium impact future' and 'high impact future'. The medium impact future scenario of air pollution emissions was also referred to as 'Most Likely Future' (MLF). The high impact future scenario had the highest emissions of air pollutants, followed by the MLF and the low impact future scenarios respectively (see Appendix E.1 for the details of each emissions scenario). The emissions inventories prepared based on these three scenarios were used to drive the TAPM-CTM model for predicting air pollution concentrations in the PPR. Given a minimal change in air pollution concentrations due to climate change between decades 1 and 2, as suggested by the results of the first project component, the project chose to use a single GCM which was the CSIRO model to predict meteorological fields for the second project component. The CSIRO model was selected because its prediction results of air quality were in the middle range of the population exposure results from the four GCMs.

For population data, two future scenarios for 2030 were developed in order to combine with the predicted air quality to assess levels of population exposure to air pollutants. The first scenario—referred to as '2030 MLF'—was based on a prediction made by the Department of Planning and Community Development Victoria. This scenario assumed

approximately 45% population growth relative to 2006 with the largest increase (~140%) in the population aged over 65 as shown in Figure 8.1. The second population scenario—referred to as '2030 high impact future'—assumed the same size of pollution (~5.9 million) as the 2030 MLF population but with a difference in spatial distribution of the population. As can be seen in Figure 8.2, the 2030 high impact assumes that half of the population growth in the outer suburbs assumed in the 2030 MLF population scenario occurs in the inner suburbs of Melbourne instead. Therefore the density of population around the centre of the PPR in the 2030 high impact future population scenario was higher than in the 2030 MLF population scenario.

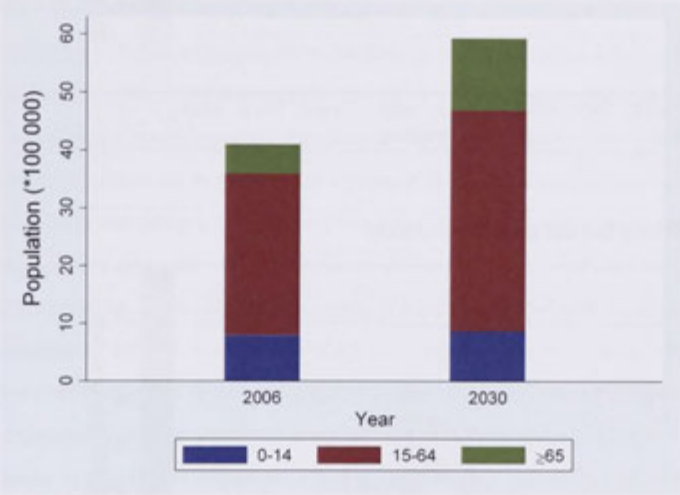
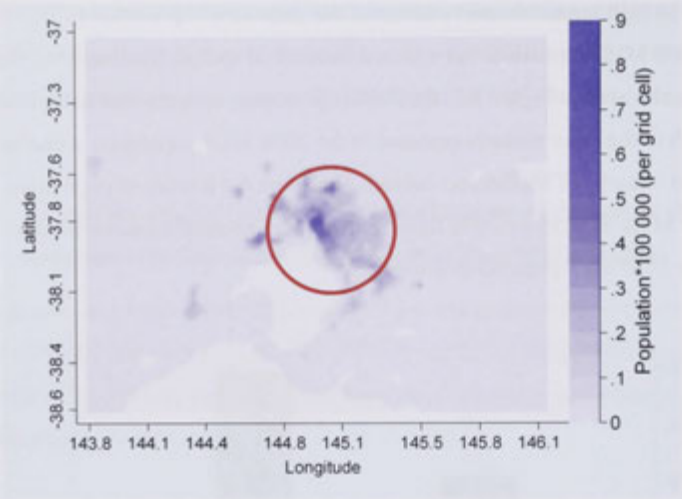


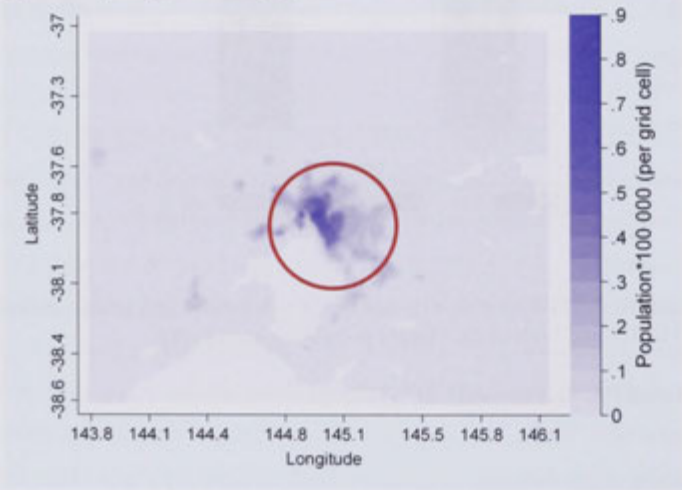
Figure 0.1 Population in the PPR stratified by age (0–14, 15–64 and ≥65 years): 2006 (census) and 2030 (MLF and high impact future population scenarios)

Source: adapted from EPA Victoria and CSIRO (2013)

(a) 2030 MLF population



(b) 2030 high impact future population



Note: The areas in the red circles are those where the population density in the high impact future scenario is higher compared to the MLF scenario.

Figure 0.2 Maps of 'static' population distribution in the PPR at 3-km grid spacing—(a) 2030 MLF and (b) 2030 high impact future scenarios.

To estimate levels of population exposure to air pollution, gridded population maps over the PPR were developed for each population scenario. These maps were referred to in the project as 'static population maps', essentially representing people at their place of residence. An extension was developed to model population movements during typical weekdays and weekends in the PPR (Walsh et al. 2011). For each of the population scenarios in each decade, two sets of twenty-four maps corresponding to the twenty-four hours of the day, one for weekdays and the other for weekend days were constructed. These maps were referred to in the project as 'dynamic population maps'.

Of the three drivers identified—climate change, air pollution emissions trends and changes in population—the impacts from climate change contributed the least to the change in air quality as well as population exposure to air pollutants between decades 1 and 2. For the combination of air pollution emissions under the 2030 MLF scenario, population under the 2030 MLF population scenario and the effect of future climate change, the CSIRO model predicted a dramatic decrease in population exposure to air pollutants primarily emitted from motor vehicles including CO, NO_2 and benzene. Without a reduction in air pollution emissions, population exposures to these primary pollutants were predicted to rise due primarily to the population growth. For example, a predicted population exposure to NO_2 was approximately $9*10^8$ ppb-person-hours/day as a result of climate change (Walsh et al. 2013). With a combination of climate change and population growth, a predicted population exposure to NO_2 was approximately $13*10^8$ ppb-person-hours/day. In contrast, such an increase in population exposure was reduced to $5*10^8$ ppb-person-hours/day when the reduction of air pollution emissions was factored in. These predictions reflect the importance of reduction in air pollution emissions to bring down the level of population exposure to primary pollutants despite the increase in future population. For $PM_{2.5}$, a reduction in population exposure of 6% between decades 1 and 2 was predicted as a result of the combined changes in climate, air pollution emissions and population assumed in the 2030 MLF scenario. However a prediction of population exposure to O_3 was in the opposite direction to the other primary pollutants and $PM_{2.5}$. The level of population exposure to O_3 max 1-h was predicted to increase by 16% between decades 1 and 2 (Walsh et al. 2013).

In addition to the combination explained above, the project constructed another two combinations of the climate change, air pollution emissions and population scenarios in order to predict corresponding changes in population exposure to the air pollutants between

the 2030s and the baseline 2000s. In these three combinations, the change in air pollution concentrations as a result of climate change was identical. The key differences were in the air pollution emissions and the population scenarios. These three combinations were i) the low impact air pollution emissions combined with the MLF population scenarios, ii) the MLF air pollution emissions combined with the MLF population scenarios and iii) the high impact air pollution emissions combined with the high impact population scenarios.

Among the three combinations of changes in climate, air pollution emissions and population, the level of population exposure to primary pollutants and $PM_{2.5}$ 24-h avg under the low impact air pollution emissions combined with the MLF population scenarios was predicted to be lower than the other two combinations. In contrast, population exposure to O_3 max 1-h under the combination of the low impact air pollution emissions and the MLF population scenarios was predicted to be higher than the other two combinations. A lower predicted level of NO_x as well as of other O_3 precursors under the low impact air pollution emissions scenario, compared to the other two scenarios of air pollution emissions, was used to explain why the predicted trend in population exposure to O_3 was in the opposite direction from the other primary pollutants and $PM_{2.5}$ (EPA Victoria & CSIRO, 2013). Note, however, that the predicted increase in average urban O_3 levels in the combined low impact air pollution emissions and the MLF population scenarios (and consequently population exposure to O_3) between decades 1 and 2 involves concentrations which are typically below the natural background. As emissions of NO_x reduce over time, the titration of O_3 levels on days of typical weather decreases. This has the effect of increasing average urban O_3 levels towards natural background levels. How this increase will affect human health is not clear, as the effect cannot be studied in isolation from the beneficial changes of reduced NO_x (Walsh et al. 2013).

8.3 Data obtained from the Future Air Projections project for estimating health impacts

Two sets of data primarily generated from the first and second components of the project were obtained for addressing Objectives 3 and 4 of this thesis respectively. Although the project made predictions for a number of air pollutants, the analyses undertaken in this chapter relied only on the simulations for $PM_{2.5}$ 24-h avg and O_3 max 8-h for consistency with the health risks estimated earlier in this thesis.

To address Objective 3, only the simulations generated by all four GCMs for decades 1 and 3 in the summer and winter months were considered. The simulations for decade 2 were not used to estimate the health impacts from climate change here due to the limitation of applying a single GCM to model the air quality. As discussed in the previous chapter, use of an ensemble of models is more scientifically appropriate in order to address model uncertainty for this type of estimation. In addition to the $PM_{2.5}$ and O_3 simulations, the 2006 gridded population data over the PPR at 3-km grid spacing were also obtained. Further, the temperature data simulated by the four GCM assemble for decades 1 and 3 were acquired for the purpose of quantifying changes in health impacts related to air pollution and climate change taking account of the temperature modifying effect.

Additional data, as listed below, generated for the second component of the project were obtained to address Objective 4 of this thesis. In each case, data were obtained from the Future Air modelling system consisting of TAPM-CTM, driven by the CSIRO model and emission scenarios supplied by the EPA Victoria.

- Predictions of temperature in a grid format over the PPR for decade 2 (the predictions of temperature for the decade 1 were the same as described above for Objective 3)
- Predictions of $PM_{2.5}$ 24-h avg and O_3 max 8-h concentrations in a grid format over the PPR for decade 2 in response to changes in climate alone (the predictions of $PM_{2.5}$ 24-h avg and O_3 max 8-h concentrations for decade 1 were the same as described above for Objective 3)
- Predictions of $PM_{2.5}$ 24-h avg and O_3 max 8-h concentrations in a grid format over the PPR for decade 2 corresponding to the three scenarios of air pollution emissions
- Maps of the 2030 population over the PPR for the MLF and the high impact future scenarios in a grid format, segregated by age groups

Note that the static population maps were used in the analyses of this chapter, rather than the dynamic population maps applied by the Future Air Projections project. The dynamic population data may be most favourable and appropriate for use in analyses where the relative health risks of air pollution are estimated based on health data collected hourly. For this thesis, the static population data are preferable as the risk estimates are based on daily counts of health endpoints.

8.4 Changes in the acute health effects of air pollution due to changes in air quality as a result of climate change

8.4.1 Methods

The methods described in this section to address Objective 3 were built upon the standard function (Equation 1.1) for estimating changes in health impacts due to exposure to an environmental hazard as stated earlier in Chapter 1.

Equation 8.1 was used to estimate changes in respiratory ED visits, the health endpoint chosen, due to exposure to air pollution under a changing climate between two time windows.

$$\Delta H = E_f - E_b \tag{0.1}$$

where ΔH is the change in respiratory ED visits (E) between a future (f) and a baseline (b) period.

Equation 8.1 was applied in this chapter to calculate changes in the number of respiratory ED visits under different scenarios and assumptions of exposure to air pollution.

8.4.1.1 Estimating the health impacts of air pollution due to climate change with no consideration of the temperature modifying effect

Estimating changes in the number of daily respiratory ED visits associated with changes in air pollution due to climate change between decades 3 and 1 was achieved by using Equation 8.2 for each decade in Equation 8.1.

$$E_d = R \sum_{i=1}^{N} \left(\frac{\sum_{t=1}^{T(d)} e^{\beta C_{i,t}(d)} - 1}{T(d)} \right) P_i \tag{0.2}$$

Equation 8.2 was applied to calculate the number of daily respiratory ED visits for each decade (d) over the domain of interest which had the total number of grid cells N. In Equation 8.2, R denotes the baseline daily rate of respiratory ED visits, β is the log relative risk for respiratory ED visits associated with a change in exposure to a certain air pollutant, $T(d)$ refers to the total number of simulation days in a decade (d), $C_{i,t}\,(d)$ is the predicted

air pollution concentration on day t in grid cell (i) in a decade (d), and P_i is the exposed population in grid cell (i).

Given that the estimation in this section focuses on the health impacts associated with changes in air pollution due to climate change alone, other parameters in Equation 8.2 including the baseline daily rate of respiratory ED visits, the log relative risk and population were assumed to be the same for the baseline (decade 1) and the future period (decade 3). According to the dose-response functions of $PM_{2.5}$ and O_3 examined in Chapter 5, there was no indication of a threshold for these two pollutants. Hence the calculations performed here assumed a zero threshold. The potential impact of threshold levels of O_3 on estimates of the health impacts attributable to air pollution and climate change was examined as part of the uncertainty assessment which is presented later in Section 8.5.

The baseline daily rate of respiratory ED visits, which excluded non-residents, was calculated as the average of daily counts of this outcome category over the period 1999 to 2008 in SD 205 divided by the population number from the 2006 census in the same geographical classification as for the daily counts. The rate calculated was specific to three summer months (December to February) and three winter months (June to August). The data on respiratory ED visits used to calculate the baseline daily rate were the same as described previously in Chapter 3. The population data were obtained from the ABS (ABS 2008). The 2006 population data were chosen as they were based on a census survey and should thus well represent the population during the mid-2000s. Furthermore, the use of 2006 population data in calculating the baseline rate of respiratory ED visits was consistent with the population data used by the Future Air Projections project to generate the gridded map of the 2006 population in the PPR. A single baseline rate of respiratory ED visits for each season was assumed to be homogeneous across the PPR domain in this analysis.

The rationale for choosing the three-month period to calculate the season-specific baseline rate of respiratory ED visits here is that it is consistent with that used to estimate the baseline log relative risks (see details in Chapters 5 and 6). Moreover, the three-month period for each season conforms to the European seasonal calendar (Wells 2013). The three-month period for each season used here was longer than the two-month period for each season used by the Future Air Projections project to predict air pollution concentrations as stated in Section 8.2.2. Nevertheless, the longer period to get more stable estimates of average daily respiratory ED visits was considered more suitable to the

context of this thesis, with the rationale for using the two-month period in the project based largely on constraints in the computational resources.

Table 8.1 contains all parameters applied in Equation 8.2 except the air pollution concentrations predicted by the four GCMs. The βs shown in Table 8.1 were taken from the estimates of respiratory ED visits associated with exposure to $PM_{2.5}$ and O_3 for all-ages presented in Chapter 5. Figure 8.3 shows a map of the 2006 population distribution over the PPR domain that is applied in Equation 8.2.

Table 0.1 Parameters used in estimating health impacts associated with air pollution due to climate change

R	summer = 3.58 per 100 000 population winter = 5.96 per 100 000 population
β* (95% CI)	$PM_{2.5}$ in summer =4.3 (0.5, 8.1) per $\mu g/m^3$ $PM_{2.5}$ in winter =1.7 (0.1, 3.2) per $\mu g/m^3$ O_3 in summer =4.5 (3.2, 5.8) per ppb
P	Population in 2006=4 114 690 in the PPR
T	decade 1=590 simulation days in summer =620 simulation days in winter decade 3=589 simulation days in summer =618 simulation days in winter
N	4 200 grid cells (70*60)

Note:*β values are shown in the scale of 10^{-3}. All the β values were estimated using a lag of 3-day moving average (lag 0-2) for both pollutants. These β values or log relative risks were converted from percentage changes in daily respiratory ED visits associated with $PM_{2.5}$ and O_3 exposure (per 10 $\mu g/m^3$ for $PM_{2.5}$ and per 10 ppb for O_3) , presented in Chapter 5, using the following equation:

log relative risk =ln ($\frac{\%\ change\ in\ daily\ respiratory\ ED\ visits}{1000}$ + 1).

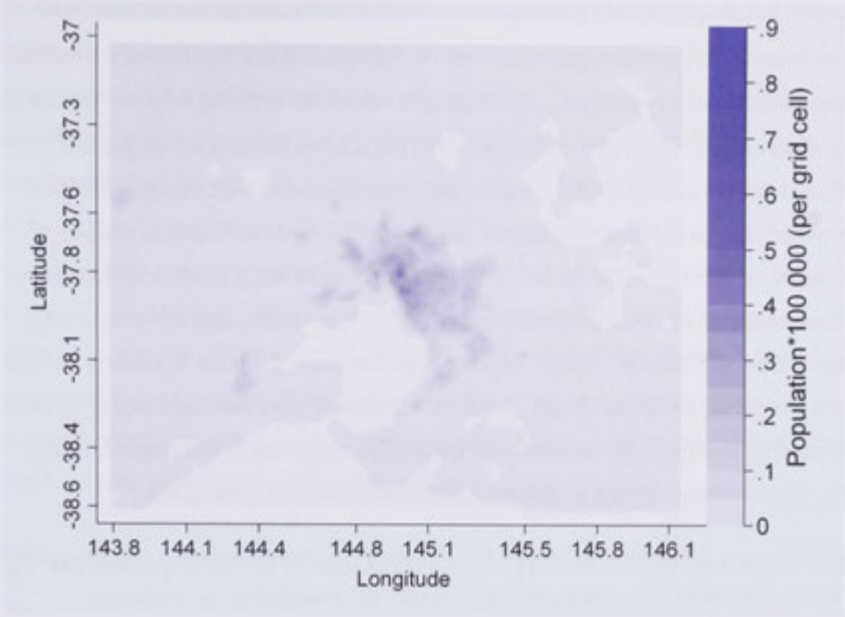


Figure 0.3 Map of 2006 population distribution in the PPR at 3-km grid spacing

8.4.1.2 Estimating the health impacts of air pollution due to climate change taking into account the temperature modifying effect

A second purpose of the analyses in this section is to estimate the health impacts associated with exposure to air pollution under a changing climate when the temperature modifying effect is taken into account. Such analyses provide information on the extent to which the interaction of temperature and air pollution affects the estimates of the health effects of air pollution, compared to estimates made by the conventional method as outlined in the previous section.

Equation 8.3 depicts the method to calculate the health impacts of air pollution due to climate change including consideration of the temperature modifying effect for each decade. Equation 8.3 was built from Equation 8.2. The notations in Equation 8.2, thus, also apply to Equation 8.3. Similar to the description above for Equation 8.2, the total change in the daily number of respiratory ED visits (taking account of the temperature modifying effect) between decades 1 and 3 was found by using Equation 8.3 in Equation 8.1.

$$E_d = R\sum_{i=1}^{N}\left(\frac{\sum_{t=1}^{T(d)} e^{\beta_{k(t)}C_{i,t}(d)} - 1}{T(d)}\right)P_i \qquad (0.3)$$

In Equation 8.3, $\beta_{k(t)}$ is the log relative risk associated with a change in exposure to air pollution in each temperature stratum as shown in Table 8.2, k is a function of t whereby the temperature on day t determines k and $\beta_{k(t)}$ is one of the three log relative risks for a given pollutant that is applicable on that day. The log relative risks shown in Table 8.2 were taken from the estimates of respiratory ED visits associated with exposure to $PM_{2.5}$ and O_3 for all-ages in the different temperature strata presented previously in Chapter 6[5]. The temperature parameter used here was the mean of maximum dry-bulb temperature on day t and minimum dry-bulb temperature on day $t-1$, as previously applied in estimating the relative risks in Chapters 5 and 6. Because exposure to air pollution is unlikely to have any protective effect on human health, negative log relative risks were assumed to be zero for this analysis. Likewise this assumption was applied elsewhere in this chapter. Apart from β_k, the parameters shown in Table 8.1 were also used for Equation 8.3.

Table 0.2 Log relative risks (95% CI) presented in the scale of 10^{-3} for respiratory ED visits associated with $PM_{2.5}$ (per $\mu g/m^3$) and O_3 (per ppb) exposure.

Pollutant	Temperature stratum			Temp cut-point (in°C)
	Low	Middle	High	
Summer				
$PM_{2.5}$	-4.3* (-11.7, 3.0)	4.3 (-0.2, 8.8)	4.8 (0.7, 8.8)	15.9, 21.5
O_3	3.7 (1.8, 5.7)	5.0 (3.6, 6.3)	3.9 (2.5, 5.3)	15.9, 21.5
Winter				
$PM_{2.5}$	3.1 (1.2, 5.1)	0.5 (-1.2, 2.2)	2.0 (-1.3, 5.3)	8.4, 11.9

Note: *This negative log relative risk was assumed to be zero when it was applied to Equations 8.3, 8.6 and 8.8.
All the log relative risks presented were estimated using a lag of 3-day moving average (lag 0-2) for both pollutants.

Simulations were performed to calculate the 95% CI for estimates of changes in the health impacts of each air pollutant resulting from climate change that involved more than one log relative risk as described in Equation 8.3. The log relative risks shown in Table 8.2 in the

[5] The percentage changes in daily respiratory ED visits associated with $PM_{2.5}$ and O_3 exposure (per 10 $\mu g/m^3$ for $PM_{2.5}$ and per 10 ppb for O_3) presented in Chapter 6 were converted to log relative risks (per $\mu g/m^3$ for $PM_{2.5}$ and per ppb for O_3) using the equation: log relative risk = $\ln(\frac{\%\ change\ in\ daily\ respiratory\ ED\ visits}{1000} + 1)$.

three temperature strata for each pollutant in each season were moderately correlated (see Appendix E.2 for the correlation matrix). Hence, sampling the three log relative risks for a given pollutant in a certain season for the three temperature strata in the simulations was undertaken using a tri-variate normal distribution. The tri-variate normal distribution was constructed from the variance-covariance matrix of the three log relative risks. For the calculation of each estimate of the changed health impacts between the two decades for a given pollutant in a certain season, the simulation was performed 1 000 times. At each sampling time, the three log relative risks for the three temperature strata were randomly sampled as a vector unit from the tri-variate normal distribution. The 95% CI for the estimated changes for a given pollutant in a certain season was derived as the 2.5^{th} percentile (lower bound) and the 97.5^{th} percentile (upper bound) of the distribution of the 1 000 estimates obtained from the simulations.

8.4.1.3 Scales used for presenting estimates of the changes in respiratory ED visits related to air pollution under future climate change scenarios

The results of the analyses estimating the effect on ED visits of changes in exposure to $PM_{2.5}$ and O_3 related to climate change alone between decades 1 and 3 that are presented later in Section 8.4.2 are expressed in four different scales: i) total change in the number of respiratory ED visits per day; ii) total change in the number of respiratory ED visits per season; iii) total change in the number of respiratory ED visits per day per head of population; and iv) percentage change in the number of respiratory ED visits per day.

The total change in the number of respiratory ED visits per day without and with taking account of the temperature modifying effect is found by using Equation 8.2 and 8.3 respectively in Equation 8.1, as described earlier. To calculate the total change in the number of respiratory ED visits per season, the daily scale was multiplied by the number of days in summer (90 days from December to February) and in winter (92 days from June to August). The rationale for using the three-month period for each season here to calculate the total change in the number of respiratory ED visits per season was the same as noted earlier in Section 8.4.1.1 for the calculation of the baseline daily rate of respiratory ED visits. Hence the predicted changes in air pollution concentrations and temperatures based on the two-month simulations for each season undertaken by the Future Air Projections project were assumed to be applicable to the estimated changes analysed for the three-month period in this chapter.

The total change in the number of respiratory ED visits per day per head of population is calculated using Equation 8.4. This equation is identical to Equation 8.1 but it is presented in the scale of the total change in the number of respiratory ED visits per day per head of population. In Equation 8.4, J_f and J_b refer to the number of respiratory ED visits per day per head of population associated with exposure to air pollutants for a future and the baseline respectively. The change in respiratory ED visits per day per head of population associated with air pollution between the two time periods is denoted as ΔI. Equations 8.5 and 8.6 are used in Equation 8.4 for the changes in the health impacts without and with considering the temperature modifying effect respectively.

$$\Delta I = J_f - J_b \tag{0.4}$$

Equation 8.5 is a modification of Equation 8.2 with an additional term of K as a denominator of P_i. This equation is used to calculate the number of respiratory ED visits per day per head of population, J for decade d. K denotes the total population over the PPR domain, in this case the 2006 population. K is calculated by summing P_i over the number of grid cells N.

$$J_d = R \sum_{i=1}^{N} \left(\frac{\sum_{t=1}^{T(d)} e^{\beta C_{i,t}(d)} - 1}{T(d)} \right) \frac{P_i}{K} \tag{0.5}$$

Similarly, Equation 8.6 is a modification of Equation 8.3 with an additional term of TP.

$$J_d = R \sum_{i=1}^{N} \left(\frac{\sum_{t=1}^{T(d)} e^{\beta_{k(t)} C_{i,t}(d)} - 1}{T(d)} \right) \frac{P_i}{K} \tag{0.6}$$

Data are presented in this scale, i.e., change in health outcome per head of population, to allow comparison of the results with other studies. In addition, this scale removes the impact of changes in population over time. For the calculations of the health impacts associated with air pollution driven by only climate change with the assumption of constant population over time, this scale does not provide additional information over that from the other scales. However, it is more informative to use this scale for assessing uncertainties associated with quantifying the health impacts due to climate change presented later in Section 8.5.

8.4.2 Results

The results in this section are presented by the types of air pollutants, $PM_{2.5}$ and O_3, in the two different seasons, summer and winter. For a given pollutant in a certain season, estimated changes in the number of respiratory ED visits due to climate change are reported for the four GCMs employed. Estimated changes in the number of respiratory ED visits associated with exposure to $PM_{2.5}$ and O_3 per day and per season between decades 1 and 3 are presented in Figure 8.4 in two different circumstances—with and without taking into account the temperature modifying effect (see Appendices E.3 and E.4 for the estimated changes presented in tabular form). The corresponding changes in the number of respiratory ED visits per day per head of population and percentage changes in the number of respiratory ED visits per day are reported in Table 8.3 and Figure 8.5 respectively.

It can be clearly seen in Figures 8.4 and 8.5 that the 95% CIs of the estimated changes between decade 1 and decade 3 in the number of respiratory ED visits associated with both $PM_{2.5}$ and O_3 for both seasons that included the temperature modifying effect were very wide. The wide CIs were largely due to the use of three separate relative risk estimates, corresponding to the three temperature strata, to estimate the changes in the health impacts. Intrinsically, a sum of multiple variances (e.g., three variances of the three separate relative risk estimates for the method considering the temperature modifying effect) to obtain a total variance is larger than that using a single variance (e.g., one variance of the single relative risk estimate for the method with no consideration of the temperature modifying effect). Hence, when the results obtained from these two methods were compared, the focus of interpretation in the following section is on the best estimate of the effect, i.e., the point estimate, rather than the 95% interval estimates. Furthermore, the intention of the comparison of the estimates of changes in the health effects of air pollution due to climate change derived from the two different methods here was not to test a null hypothesis, i.e., testing to indicate the presence (or not) of a statistically significant difference, as is commonly applied when comparing two independent estimates. Rather, the description of the differences between the two sets of estimated changes using the two different methods, which are really estimates of the same effect, is focused on illustrating a potential trend for the differences in their direction and magnitude.

8.4.2.1 $PM_{2.5}$ in summer

As mentioned in Section 8.2.2, the four GCM models predicted an increase in population exposure to $PM_{2.5}$ concentrations in the summer between the baseline and the 2070s. As a result, using the conventional method (in the absence of any temperature modifying effect), there was an estimated increase in the number of respiratory ED visits, ranging from 0.09 (95% CI: 0.01, 0.18) to 0.31 (95% CI: 0.03, 0.60) additional ED visits per day (8 (95% CI: 1, 16) to 28 (95% CI: 3, 54) per season) (see Figure 8.4a). These estimated changes correspond to a range of 0.022 (95% CI: 0.002, 0.043) to 0.075 (95% CI: 0.008, 0.146) excess ED visits per million population per day (see Table 8.3).

Overall, the point estimates from the analyses of the effect on respiratory ED visits of changes in exposure to $PM_{2.5}$ in summer that included the temperature modifying effect were higher than the changes predicted using the conventional method. It can be seen from Figure 8.5a that, among the four GCM modelling systems, the largest difference (~12%) in percentage change in daily respiratory ED visits between these two methods was for the analyses using data predicted from the GFDL model. The interval estimates predicted by the four GCMs, both absolute and relative terms as shown in Figures 8.4a and 8.5a respectively, using the alternative method considering the temperature modifying effect, suggested the greater plausibility of an increase in the number of $PM_{2.5}$-related respiratory ED visits in the summer between the two decades. This is exhibited by the majority of the predicted ranges that fall into the positive changes.

Consideration of the difference between the two decades and for each season of the number of days that lie within each temperature stratum (see Figure 8.6) provides some explanation of why taking account of the temperature modifying effect increases the magnitude of the changes in respiratory ED visits associated with $PM_{2.5}$ in the summer. It can be seen that warmer temperatures in the summer, predicted in decade 3 under climate change, resulted in an increase in the number of days in the high temperature stratum ($\geq$21.5°C) and a decrease in the number of days in the low temperature stratum (<15.9°C), compared to decade 1. Taken together the stronger risk estimate associated with $PM_{2.5}$ exposure in the summer in the high temperature stratum as presented in Table 8.2 and the increase in the number of days in this temperature stratum in decade 3, therefore, led to a rise in the estimated changes in respiratory ED visits relative to when the constant risk estimate associated with $PM_{2.5}$ across the temperature range was applied.

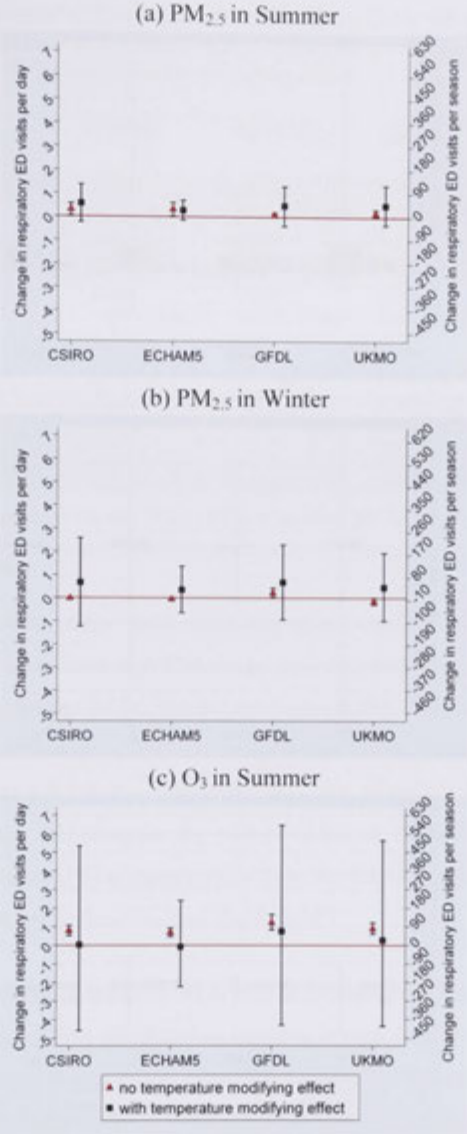


Figure 0.4 Estimated changes (95% CI) in respiratory ED visits per day and per season between decades 1 and 3 associated with exposure to $PM_{2.5}$ and O_3 due to climate change: with and without taking into consideration the temperature modifying effect

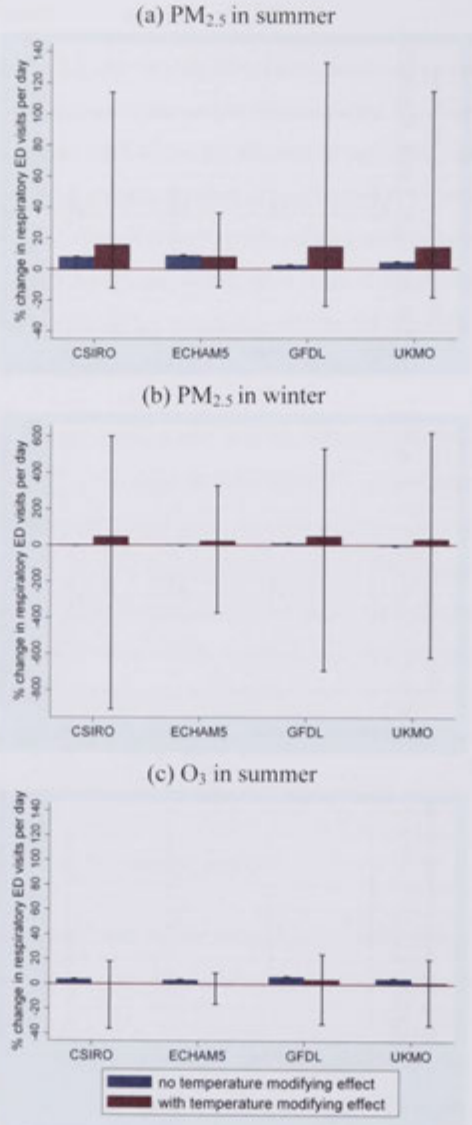


Figure 0.5 Percentage changes (95% CI) between decades 1 and 3 in daily respiratory ED visits associated with exposure to $PM_{2.5}$ and O_3: with and without taking account of the temperature modifying effect

Table 0.3 Estimated changes* (95% CI) in daily respiratory ED visits per head of population between decades 1 and 3 associated with exposure to $PM_{2.5}$ and O_3: with and without taking account of the temperature modifying effect

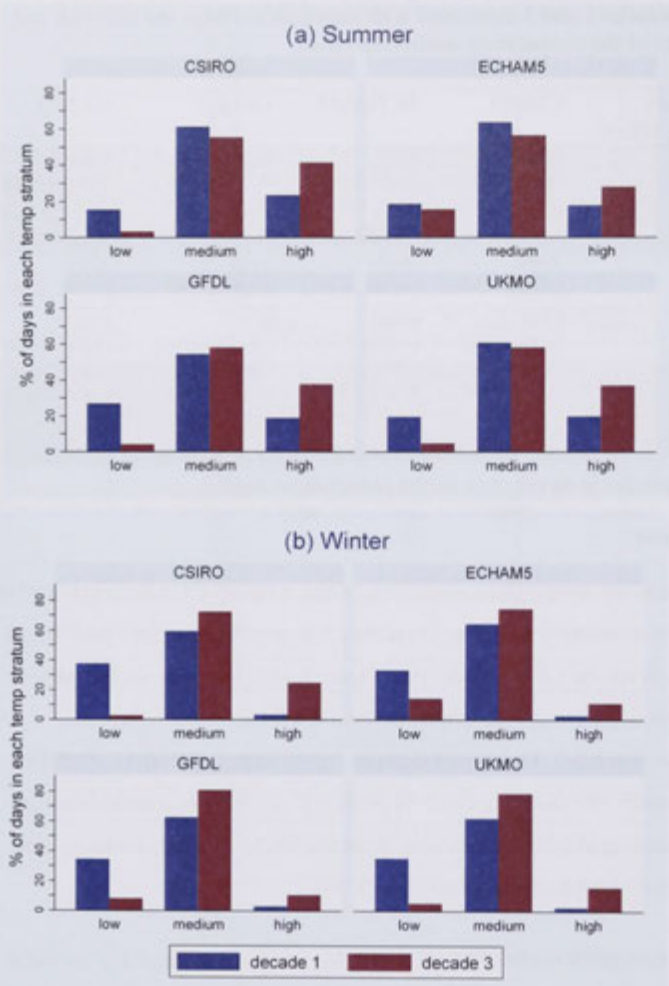
Pollutant	Temperature modifying effect	CSIRO	ECHAM5	GFDL	UKMO
$PM_{2.5}$ Summer	No	0.069 (0.008, 0.135)	0.075 (0.008, 0.146)	0.022 (0.002, 0.043)	0.040 (0.004, 0.080)
	Yes	0.138 (-0.060, 0.333)	0.067 (-0.034, 0.169)	0.120 (-0.093, 0.315)	0.125 (-0.082, 0.332)
$PM_{2.5}$ Winter	No	-0.006 (-0.011, 0.000)	-0.259 (-0.026, -0.001)	0.053 (0.004, 0.104)	-0.036 (-0.070, -0.003)
	Yes	0.162 (-2.999, 0.628)	0.085 (-0.156, 0.334)	0.165 (-0.227, 0.558)	0.111 (-0.243, 0.473)
O_3 Summer	No	1.912 (1.287, 2.597)	1.648 (1.111, 2.233)	2.969 (2.007, 4.014)	2.169 (1.450, 2.965)
	Yes	0.008 (-1.114, 1.297)	-0.222 (-0.543, 0.586)	0.183 (-1.046, 1.567)	0.066 (-1.063, 1.371)

Note: *The changes are presented in the unit of per million population per day.

8.4.2.2 $PM_{2.5}$ in winter

With no consideration of the temperature modifying effect, a reduction in the total number of respiratory ED visits associated with $PM_{2.5}$ exposure was predicted in the winter by all of the GCM models, except the GFDL model (see Figure 8.4b). The cause of the estimate of increased population exposure to $PM_{2.5}$ in winter by the GFDL model only was explained earlier in Section 8.2.2. The largest decrease of 0.15 (95% CI: -0.01, -0.29) in total number of respiratory ED visits per day (-14 (95% CI: -1, -27) per season) between the two decades was estimated by the predictions from the UKMO model, corresponding to a 6.6 % (95% CI: -6.6, -6.6) decrease (see Figure 8.5b).

On the contrary, the predictions made by all of the GCMs using the method that included the temperature modifying effect suggested an increase in total number of respiratory ED visits under climate change. Based on the predictions for the CSIRO and GFDL models, a change in total number of respiratory ED visits per day was estimated to be higher at 0.67 (95% CI:-1.23, 2.59) and 0.68 (95% CI: -0.94, 2.30) respectively (corresponding to an additional 62 (95% CI: -113, 238) and 62 (95% CI: -86, 211) ED visits per season) in decade 3, compared to the baseline level. The predictions derived from the ECHAM5 and UKMO models for the change in total number of respiratory ED visits in association with $PM_{2.5}$ in winter were slightly lower: an increase of 0.35 (95% CI: -0.64, 1.37) and 0.46 (95% CI: -1.00, 1.95), per day respectively (corresponding to 32 (95% CI: -59, 126) and 42 (95% CI: -92, 179) additional respiratory ED visits per season).



Note: The cut-points used for stratifying the temperature data in the summer were 15.9°C and 21.5°C. In winter, the strata were divided by using the temperature cut-points of 8.4°C and 11.9°C.

Figure 0.6 Percentage of days in each temperature stratum in decades 1 and 3 in the summer and winter

The greater total number of respiratory ED visits associated with $PM_{2.5}$ in the winter when taking account of the temperature modifying effect was unexpected. It would be more reasonable to expect a decrease in total number of respiratory ED visits between the two decades for two reasons. Firstly, there was a dramatic decrease in the number of days in the low temperature stratum predicted by all of the four GCMs. For example, there was a 35% decrease in the number of days in the low temperature stratum (<8.4°C) predicted by the CSIRO model between decades 1 and 3 in the winter (see Figure 8.6b). Secondly, the strongest log relative risk associated with $PM_{2.5}$ exposure (point estimate of $3.1*10^{-3}$) was estimated for the low temperature stratum in winter, compared to the log relative risks in the other two temperature strata.

The explanation for the finding of excess $PM_{2.5}$-related respiratory ED visits in the winter may lie in the following observations. Firstly, the warmer climate predicted by all the four GCMs in decade 3 not only lowered the number of days in the low temperature stratum as described above, but it also increased the number of days in the high temperature stratum. For example, the number of days predicted by the CSIRO model in the high temperature stratum in decade 3 was 21.5% higher than that in decade 1. Secondly, the increased number of days in decade 3 in this temperature stratum together with the log relative risk estimated for this stratum (point estimate of $2.0*10^{-3}$) that was slightly weaker than that estimated in the low temperature stratum could explain the additional respiratory ED visits. Thirdly, as shown in Table 8.4, except for the UKMO model, $PM_{2.5}$ concentrations in the high temperature stratum were predicted by all the GCMs to be higher in decade 3 compared to decade 1. Further, the magnitude of the increased $PM_{2.5}$ concentrations in the high temperature stratum was greater than the reduced $PM_{2.5}$ concentrations estimated for the low temperature stratum. Taken together, these factors affecting the high temperature stratum led to the estimate of excess $PM_{2.5}$-related respiratory ED visits for this temperature stratum between decades 3 and 1. The greater magnitude of this estimate of excess ED visits outweighed the reduction in respiratory ED visits estimated for the low temperature stratum, leading to an overall estimate of additional health impact for decade 3 compared to decade 1.

Table 0.4 Change (percentage change) in $PM_{2.5}$ concentrations ($\mu g/m^3$) in each temperature stratum in the winter between decades 1 and 3

GCM	Temperature stratum*		
	Low	Medium	High
CSIRO	-0.06 (-2.3%)	0.16 (5.9%)	0.08 (3.4%)
ECHAM5	-0.00 (-0.0%)	-0.01 (-0.3%)	0.06 (2.5%)
GFDL	-0.01 (-0.0%)	0.28 (10.6%)	0.45 (20.7%)
UKMO	-0.22 (-8.2%)	-0.12 (-4.2%)	-0.08 (-3.4%)

Note: The temperature strata were divided by using the temperature cut-points of 8.4°C and 11.9°C.

The greater $PM_{2.5}$ concentrations in the high temperature stratum in winter predicted by the CSIRO, ECHAM5 and GFDL models may be due to an increase in ambient concentrations of the sea salt component of $PM_{2.5}$ in the winter that is likely to occur on warm days. To support this, Figure 8.7 shows that the magnitude of the excess $PM_{2.5}$-related respiratory ED visits in decade 1 compared to decade 3 as predicted by the CSIRO model where the temperature modifying effect was included, was most apparent around the Port Phillip Bay compared to other areas. The results presented here are consistent with an explanation noted by the Future Air Projections project about a spatial variation of relative changes between decades 1 and 3 in population exposure to $PM_{2.5}$ in winter (Cope et al. 2011c). Cope et al.(2011c) noted that there were reductions of population exposure to $PM_{2.5}$ in winter that were predicted by the CSIRO, ECHAM5, and UKMO models in most areas of Melbourne. However, in the areas around the bay, there was a predicted increase in population exposure to $PM_{2.5}$. It was further noted that this was probably related to stronger winds around the bay on warm days, compared to cold days, resulting in strong waves and increased sea salt aerosols. The estimate of increased $PM_{2.5}$ -related respiratory ED visits per day around the bay was also observed based on the predictions of the other GCMs with varying magnitude (see Appendix E.5).

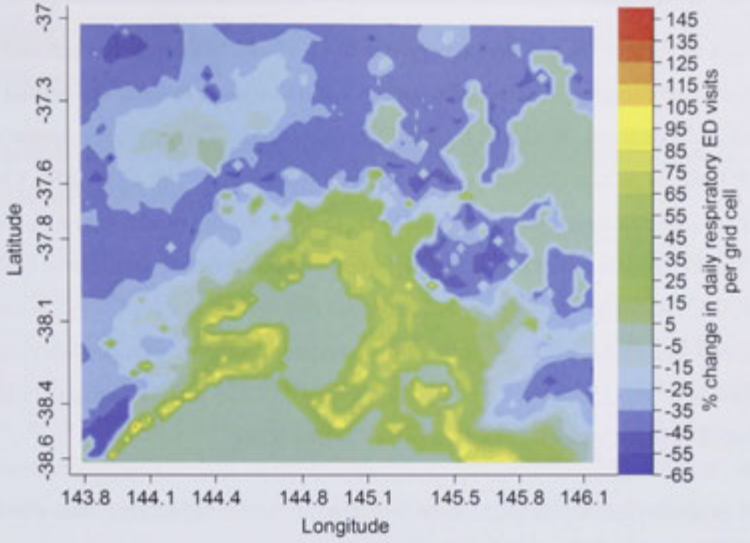


Figure 0.7 Map of percentage changes in the number of daily respiratory ED visits in winter predicted by the CSIRO model between decades 1 and 3 in the PPR using the method taking account of the temperature modifying effect

Although this is a possible explanation for the finding of increased $PM_{2.5}$-related respiratory ED visits in winter between the two decades when the temperature modifying effect is included, it is also possible that the results have occurred by chance. The widest 95% CIs of the estimated percentage changes in $PM_{2.5}$-related respiratory ED visits in the winter comparative to those for $PM_{2.5}$ and O_3 in the summer, as shown in Figures 8.5, indicated considerable uncertainty in the estimates, partly due to the application of the non-significant log relative risks estimated for the middle and high temperature strata in the analyses. Moreover, despite the increased predictions, the 95% CI estimates of the percentage changes in $PM_{2.5}$-related respiratory ED visits in the winter span almost equally across both positive and negative changes. Perhaps, this also suggested low precision of the predicted increases, based on the best point estimates, in $PM_{2.5}$-related respiratory ED visits in the winter between the two decades. Future studies are required to validate the results here, especially those that consider the influence of the temperature modifying effect on the health effects of $PM_{2.5}$ exposure in the winter and how it plays out in the context of climate change.

8.4.2.3 O_3 in summer

In line with an increase in population exposure to O_3 concentrations in the summer between the two decades, as noted in Section 8.2.2, an increase in daily O_3-related respiratory ED visits was predicted by all of the four GCMs. The greatest increase of 1.22 (95% CI: 0.83, 1.65) in total number of respiratory ED visits per day (110 (95% CI: 74, 149) per season) associated with exposure to O_3 in the summer was predicted by the GFDL model. The ECHAM5 model predicted the lowest increase, with an estimate of 0.68 (95% CI: 0.46, 0.92) additional respiratory ED visits per day (61 (95% CI: 41, 83) per season) (see Figure 8.4c). Presented in the scale of percentage change, the changes reported here correspond to a 5.8% (95% CI: 5.7, 6.0) and 3.2% (95% CI: 3.1, 3.3) increase for the GFDL and ECHAM5 models respectively (see Figure 8.5c).

Figure 8.4c shows that accounting for the temperature modifying effect resulted in little change to the estimates. These point estimates for the change in respiratory ED visits were lower, for all of the GCM models, compared to when the temperature modifying effect was disregarded. For example, using the predictions based on the CSIRO model, an additional 0.79 (95% CI: 0.53, 1.07) total number of respiratory ED visits per day between decades 1 and 3 using the conventional method reduced to an increase of 0.03 (95% CI: -4.59, 5.34) when the temperature modifying effects were included.

It is difficult to discern why the difference in the estimates of total number of respiratory ED visits related to O_3 in summer per day between the two decades when taking account of the temperature modifying effect is only small. However such minimal changes between the two decades may be primarily explained by the pattern of relative risks in the different temperature strata. As presented in Table 8.2, the log relative risk for respiratory ED visits in the middle temperature stratum (point estimate of $5.0*10^{-3}$, 15.9–21.5°C) was the highest, followed by that in the high (point estimate of $3.9*10^{-3}$, >21.5°C) and low (point estimate of $3.7*10^{-3}$, <15.9°C) temperature strata respectively. Since there was little change between the decades in the number of summer days in the middle temperature stratum where the strongest risk associated with O_3 was identified, overall this led to little change in respiratory ED visits. It might be argued that from decades 1 to 3, there was an increase in the number of days in the high temperature stratum (for example an 18% increase based on the CSIRO model). However because of the weaker risk estimates (point estimate of $3.9*10^{-3}$) related to O_3 exposure in the high temperature stratum, the impact of

the higher number of days in this temperature stratum in decade 3 compared to decade 1 was balanced by the decreased number of days in the low temperature stratum that had a similar risk estimate.

8.5 Assessment of uncertainties associated with quantifying health impacts attributed to future air quality

To assess uncertainties related to health impacts attributed to future air quality under climate change, a number of combinations of factors were constructed based on identified key uncertainties which were likely to influence the estimations. As mentioned in Section 8.2.3, the Future Air Projections project identified three key drivers affecting the change in future air quality. These drivers were climate change, changes in air pollution emissions and changes in population size, age structure and distribution. Building on the drivers identified by the project, the sensitivity of the estimates of health impacts to the uncertainties within three groups of factor(s), referred to henceforth as 'Group A', 'Group B', and 'Group C' were assessed. There were three sub-groups within Group C—'Group C1', 'Group C2' and 'Group C3'—corresponding to the low impact, MLF and high impact air pollution emissions scenarios developed by the project.

Based on Equation 1.1 that is generally used in estimating health impacts due to exposure to an environmental factor, it can be seen that the project focused on two contributing variables—changes in air pollution concentrations (ΔC) and population (P). Thus the emphasis of this section of the thesis, which adds to the work of the project, is in the examination of the effect of uncertainties associated with the relative health risks of exposure to air pollution (β) on estimations of the health effects of future air pollution. Apart from Groups A to C, another three groups were also assessed to investigate uncertainties associated with i) the temperature modifying effect, ii) the age distribution and choice of dose-response functions and iii) threshold levels of O_3 (see Table 8.5). To be consistent with naming Groups A to C, the latter three groups are referred to from this point forward as 'Group D', 'Group E' and 'Group F' respectively. Groups E and F had four and three sub-groups respectively and these sub-groups are referred to by adding a numerical sequence starting from 1 to the name of each group, such as 'Group E1' to 'Group E4'.

Table 0.5 Groups of factor(s) used to assess the sensitivity of the estimates to uncertainties in the estimation of changes in respiratory morbidity associated with future air pollution exposure

A	Climate change only
B	Climate change + population growth (2030)
C	Climate change + population growth (2030) + air pollution emissions (C1–low impact, C2–MLF and C3–high impact)
D	Climate change + population growth (2030) + air pollution emissions (MLF) + temperature modifying effect
E	Climate change + population growth (2030) + air pollution emissions (MLF) + age distribution + the choice of dose-response functions (E1 to E4)
F	Climate change + population growth (2030) + air pollution emissions (MLF) + O_3 threshold levels (F1 to F3)

Groups D, E and F were examined by combining the uncertainty identified for each of these groups on top of Group C2—a combination of climate change, the MLF air pollution emissions and the MLF population scenario. The rationale was that Group C2 was representative of the mid-range future driven by the most plausible scenarios of the three critical factors fundamentally determining air quality in the 2030s.

The selection of each factor to be investigated under Groups D, E and F was justified on the basis of the magnitude and importance of uncertainties suggested by past studies, particularly at the stage of health impact projections as detailed in Chapter 7. The rationale for choosing these factors is briefly reiterated below.

- Group D (temperature modifying effect)

As can be seen from Section 8.4.2, taking into account the temperature modifying effect can potentially alter estimations of the health impacts associated with air pollution under climate change. An assessment of the effect of taking account of the temperature modifying effect was repeated here, this time for decade 2, under this group so that the results of changes in the health endpoint of interest could be compared to those derived from the other groups.

- Group E (age distribution and the choice of dose-response functions)

As for other developed countries, the demographic structure in Australia is changing towards an ageing population. As reviewed in Chapter 2, the elderly are identified in a number of studies as a high-risk population with regard to health risks of exposure to air

pollution. As a result, population ageing predicted in the future may lead to an increase in the health impacts of air pollution, despite improved air quality.

This group, hence, first examined the impact of age distribution by using relative risks associated with the pollutants of concern estimated specifically for three age groups (0–14, 15–64 and ≥65 years) to quantify changes in future respiratory morbidity. The three relative risks were estimated by using the blending approach that was also used to estimate the log relative risks for all-ages applied under Groups A, B, C, D and F.

A few studies have reported the uncertainties due to the choice of dose-response functions for estimating the impact of climate change on future health effects of air pollution (Bell et al. 2007a). Bell et al. (2007a) reported that a percentage increase estimated for the US (average for 50 cities) in O_3-related non-accidental mortality due to climate change between 1990s and 2050s varied from 0.11% (95% CI: 0.005, 0.21) to 0.27% (95% CI: 0.16, 0.38) when using dose-response functions derived from different epidemiological studies. Selin et al. (2009) reported that when considering uncertainty in dose-response function and the method for economic valuation, an estimate of the difference in total global loss in economic welfare due to O_3-related health impacts driven by climate and emission changes between 2000 and 2050 varied from 13 to 190 million dollars for a 95% probability interval. Indeed, the choice of dose-response functions may be more important than the choice of climate models. Thus, another three sets of relative risks were compared under this group to explore the uncertainty due to the choice of dose-response functions. More details about the rationale for choosing these specific relative risks are given later in Section 8.5.1.1. Of the three sets of relative risks, two sets that were derived from other studies had only age-specific relative risks (with no relative risks for all-ages). Therefore, assessment of the choice of dose-response functions was undertaken in combination with the assessment of uncertainty associated with the age structure.

To make the uncertainty analysis consistent across all of the four sub-groups within Group E, relative risks for respiratory ED visits derived from the other two studies had to be available for age-specific (three age groups), season-specific (two seasons) and both pollutants ($PM_{2.5}$ and O_3). Unfortunately, none of the previous time-series and case-crossover studies in Australia (as reviewed in Chapter 2) provided the relative risks required for the analysis under this group. The only available relative risks from past studies that met the specified requirements were for the outcome 'respiratory emergency

hospital admissions'. Therefore, the analyses for comparing the choice of dose-response functions derived from other studies were conducted using relative risks for respiratory emergency hospital admissions, rather than for respiratory ED visits.

'ED visits' is generally considered to be a milder form of morbidity than 'emergency hospital admissions'. Due to the ambient levels of air pollutants in Australia, that are generally below the required standards, exposure to ambient air pollution is likely to be more closely associated with a weaker form of morbidity, e.g., ED visits, resulting in a stronger relative risk than that for hospital admissions. However, results of a study by Winquist et al. (2012) suggested that differences between relative risks associated with exposure to air pollutants for ED visits and for hospital admissions depended upon the pollutant, health outcome and age of interest. For respiratory outcomes, that study reported that a relative risk for ED visits related to $PM_{2.5}$ and O_3 exposure was stronger than that for emergency hospital admissions only for young ages (<18 years). The magnitude of a relative risk for ED visits for a respiratory outcome resulting from $PM_{2.5}$ and O_3 exposure for the 19–64 years age group as well as for the elderly aged above 65 years was similar to that for patients hospitalised.

Given the above, in the context of the analysis for Group E, the use of relative risks for hospital admissions instead of for ED visits is likely to provide a reasonable estimation of changes in the number of respiratory ED visits under Group E. This is due to the comparable magnitude of a relative risk associated with $PM_{2.5}$ and O_3 exposure for respiratory ED visits and for respiratory hospital admissions for the elderly; the age group predicted to have the highest growth in 2030 compared to the other two age groups. Furthermore, the general purpose of the analyses conducted under this section of this chapter is to be more representative of the sensitivity of future changes in air pollution-related respiratory morbidity due to non-climate factors, rather than being specific to respiratory ED visits or respiratory hospital admissions. Hence the use of relative risks for hospital admissions in place of those for respiratory ED visits lies within the scope of Objective 4 addressed under this section.

- Group F (O_3 threshold level)

Previous studies quantifying health impacts associated with changes in O_3 driven by climate change have variously applied an O_3 threshold. Most studies which applied a

threshold assumed only a single level. This group, hence, was examined by making use of varying O_3 threshold levels. Three thresholds including 10, 20 and 30 ppb (with a zero threshold applied under the other groups), were tested. The selection of these three threshold concentrations was based on evidence derived from a number of studies that suggested that if a O_3 threshold effect exists, it would be approximately within this range of O_3 max 8-h concentrations (Bell et al. 2006; Stylianou and Nicolich 2009; Kim et al. 2004).

8.5.1 Methods

8.5.1.1 Methods to assess the uncertainty

As described in Section 8.3, the data used in the analyses under this section were mainly the research outputs of the second component of the Future Air Projections project. Similar to the analyses conducted in Section 8.4, the fundamental method here was to calculate estimates of changes in respiratory morbidity between decades 1 and 2 as described in Equation 8.1.

Apart from Group A in which only the impact of climate change was examined, and Equation 8.2 was applied, the calculations undertaken are described below for each group.

- Groups B and C

$$E_d = R \sum_{i=1}^{N} \left(\frac{\sum_{t=1}^{T(d)} e^{\beta C_{i,t}(d)} - 1}{T(d)} \right) P_i(d) \qquad (0.7)$$

Equation 8.7 builds on Equation 8.2. The only difference between these two equations was the use of a specific population size and distribution for each decade d. For decade 1, the 2006 population scenario was employed in Equation 8.7. For decade 2, the 2030 MLF population scenario was applied for Groups B, C1 and C2, whereas the 2030 high impact population scenario was applied for Group C3.

Air pollution concentrations used in Group B for decade 2 were the same as those applied in Equation 8.2 but limited to predictions from the CSIRO model driven by the 2006 emissions inventory. In Group C, air pollution concentrations for decade 2 were predicted based on the three scenarios of air pollution emissions as described earlier.

- Group D (temperature modifying effect)

$$E_d = R\sum_{i=1}^{N}\left(\frac{\sum_{t=1}^{T(d)} e^{\beta_{k(t)}C_{i,t}(d)}-1}{T(d)}\right)P_i(d) \qquad (0.8)$$

Equation 8.8 builds on Equation 8.3. Similar to that described above for Equation 8.7, the only difference was the use of a specific population size and distribution for each decade d, thus allowing a study of changes to population whilst including the temperature modifying effect. The 2006 and the 2030 MLF population scenarios were used in Equation 8.8 for decades 1 and 2 respectively.

- Group E (age distribution and the choice of dose-response functions)

$$E_d = \sum_{j=1,2,3} R_j \sum_{i=1}^{N}\left(\frac{\sum_{t=1}^{T(d)} e^{\beta_j C_{i,t}(d)}-1}{T(d)}\right)P_{i,j}(d) \qquad (0.9)$$

In Equation 8.9, instead of using a single baseline daily rate of respiratory ED visits for all-ages, different rates corresponding to three different age groups j (0–14, 15–64 and ≥65 years) as shown in Table 8.6 were applied. For β_j and $P_{i,j}$, similarly, a specific log relative risk and population for each age group j was applied in each grid cell (i). Table 8.7 presents population in each age group in decades 1 and 2 as applied here. Table 8.8 contains the estimates of β_j applied in this equation.

The calculation of the baseline daily rate of respiratory ED visits for each age group presented in Table 8.6 was similar to that described in Section 8.4.1.1 except that the rate is specific to each age group for each season. Note that when the calculation using this equation involved an estimate of change in $PM_{2.5}$- and O_3-related *respiratory hospital admissions* between decades 1 and 2, it was assumed that the age-specific baseline daily rates of *respiratory hospital admissions* were equivalent to those for *respiratory ED visits* as shown in Table 8.6.

Under this group, four sets of dose-response functions (i.e., log relative risks) estimated from different epidemiological approaches were examined (referred to here as Groups E1 to E4). The first set of log relative risks applied under Group E1 was derived from applying the blending approach presented in Chapter 5. The second set of log relative risks applied under Group E2 was derived from applying the standard approach as presented in Chapter 5.

The other two sets of log relative risks used under this group were derived from two external studies. The third set of log relative risks was from the EPHC study which applied a multi-city case-crossover approach to estimate health risks, including those for emergency hospital admissions, associated with air pollution in major cities of Australia during 1998 to 2001 (Environmental Protection and Heritage Council 2010). The EPHC study divided the respiratory admissions data by age into five categories (0, 1–4, 5–14, 15–64 and ≥65 years) for the purpose of seasonal analysis of increased risks for respiratory admissions associated with exposure to air pollution. In the young population aged 0–14 years, due to the difference in age categorisation between this section of the thesis and the EPHC study, only log relative risks for the 1–4 years age group of the EPHC study were used here. The rationale was that this age group had the highest rate for respiratory admissions among the three young age groups.

The fourth set of log relative risks was taken from a study conducted by EPA Victoria to investigate the impacts of air pollution on hospital admissions during 1994 to 1997 in Melbourne by using a time-series approach (EPA Victoria 2001). The time-series applied to the study by EPA Victoria was slightly different from those applied to the blending and standard approaches in terms of types of smoothers to control for time trends and meteorological variables. Note that despite using the time-series in the blending and standard approaches in this thesis as well as the study by EPA Victoria, as shown in Table 8.8, the lag times applied to these approaches were different.

Table 0.6 Baseline daily rate of respiratory ED visits per 100 000 population for each age group for the summer and the winter

Age	Summer	Winter
0–14	8.01	16.65
15–64	1.85	2.41
≥65	6.61	9.75

Table 0.7 Age-group specific population in decades 1 and 2

Age	Decade 1	Decade 2
0-14	791 273	871 552
15–64	2 814 387	3 841 534
≥65	509 030	1 240 286

Table 0.8 Estimated log relative risks (β) expressed in the scale of 10^{-3} (95% CI) for respiratory ED visits/hospital admissions associated with exposure to $PM_{2.5}$ (per $\mu g/m^3$) and O_3 (per ppb) in three age groups.

	Group E1[a]	Lag	Group E2[b]	Lag	Group E3[c]	Lag	Group E4[d]	Lag
$PM_{2.5}$ in summer								
0–14	4.3 (-1.7, 10.4)	3-D avg	3.1 (-3.4, 9.6)	3-D avg	7.9 (2.1, 13.7)	2-D avg	10.9 (-5.6, 31.4)	5-D avg
15–64	1.1 (-5.2, 7.4)	3-D avg	2.9 (-3.8, 9.6)	3-D avg	-0.5* (-10.1, 9.5)	2-D avg	3.7 (-7.1, 16.4)	lag2
≥65	8.6 (1.0, 16.3)	3-D avg	6.8 (-1.3, 14.8)	3-D avg	1.9 (-2.1, 6.1)	2-D avg	14.9 (3.4, 28.0)	lag1
$PM_{2.5}$ in winter								
0–14	3.1 (0.9, 5.2)	3-D avg	2.0 (-0.2, 4.1)	3-D avg	2.9 (-3.0, 6.1)	2-D avg	-0.8* (-4.0, 2.5)	lag2
15–64	-0.5* (-3.5, 2.5)	3-D avg	0.4 (-2.5, 3.4)	3-D avg	3.4 (0.8, 5.8)	2-D avg	5.6 (0.8, 10.6)	3-D avg
≥65	1.3 (-2.1, 4.7)	3-D avg	2.5 (0.0, 3.2)	3-D avg	3.7 (1.1, 6.6)	2-D avg	4.5 (-5.0, 9.8)	5-D avg
O_3 in summer								
0–14	4.5 (2.5, 6.5)	3-D avg	3.4 (1.2, 5.6)	3-D avg	6.0 (3.7, 8.2)	2-D avg	5.0 (1.3, 8.8)	5-D avg
15–64	4.6 (2.4, 6.8)	3-D avg	4.1 (1.7, 6.5)	3-D avg	1.2 (-2.2, 4.7)	2-D avg	2.1 (-4.0, 4.7)	5-D avg
≥65	4.4 (1.7, 7.1)	3-D avg	2.7 (-0.3, 5.7)	3-D avg	0.5 (-2.4, 3.4)	2-D avg	2.5 (0.7, 4.3)	5-D avg

Note: *these negative log relative risks were assumed to be zero when they were applied to Equation 8.9.

[a] Risk estimates for Group E1 (respiratory ED visits) were derived from the blending approach presented in Chapter 5.

[b] Risk estimates for Group E2 (respiratory ED visits) were derived from the standard approach presented in Chapter 5.

[c] Risk estimates for Group E3 (respiratory hospital admissions) were derived from the EPHC Study (Environmental Protection and Heritage Council 2010).

[d] Risk estimates for Group E4 (respiratory hospital admissions) were derived from a study conducted by EPA Victoria (EPA Victoria 2001).

Since calculations of the number of daily respiratory morbidity outcomes (ED visits and emergency hospital admissions) and estimates of the changes between the two decades under this group involved using three log relative risks corresponding to the three different age groups for a given pollutant and season at a time, as explained above, simulations were used to obtain 95% CIs. The log relative risks applied under Groups E1 and E2 for the three age groups for a given pollutant in a particular season were assumed to be uncorrelated. This assumption was based on the way that the statistical model was fitted for each age group to estimate these log relative risks (see details in Chapter 5). For the log relative risks applied under Groups E3 and E4 that were derived from other studies, the authors provided only a general statement that their analyses were stratified by age group. Thus, it was assumed here for the purpose of calculating the 95% CIs that the log relative risks estimated for the different age groups for each pollutant in a certain season under Groups E3 and E4 were also uncorrelated. Given the uncorrelated log relative risks among the three age groups, these log relative risks could be assumed to have normal distributions. As a result, they were sampled independently in the simulations at each sampling time from their corresponding normal distributions. The normal distribution for each log relative risk for each age group had the mean equal to the estimated log relative risk itself and the standard deviation equal to the standard error of that corresponding estimated log relative risk. Other details for the simulations performed for Group E are the same as described in Section 8.4.1.2 for estimating changes in the health impacts of air pollution due to climate change between decades 1 and 3 taking account of the temperature modifying effect.

- Group F (O_3 threshold level)

$$E_d = R \sum_{i=1}^{N} \left(\frac{\sum_{t=1}^{T(d)} e^{\beta_l (C_{i,t}(d) - l)^+}}{T(d)} \right) P_i(d) \qquad (0.10)$$

Equation 8.10 is similar to Equation 8.7: with the addition of an O_3 threshold (l). There were three threshold levels tested, apart from the zero threshold level assumed earlier, including 10, 20 and 30 ppb. In grid cell i on day t for each decade (d), the threshold was first subtracted from the concentration of air pollution before being multiplied with the log relative risk specific to each threshold level, β_l. If the concentration after subtraction was below zero, it was set to be zero.

The log relative risks as shown in Table 8.9 were applied for this group. They were derived from refitting Model 5.3 presented in Chapter 5, replacing O_3 concentrations with those above each corresponding threshold. For example, to estimate a log relative risk for respiratory ED visits associated with O_3 exposure for the 10 ppb threshold, on a given day, the 10 ppb was subtracted from the O_3 concentration. If the concentration after subtraction was below zero, it was taken to be a zero concentration.

Table 0.9 Estimated log relative risks ($\boldsymbol{\beta}$) expressed on the scale of 10^{-3} (95% CI) for respiratory ED visits associated with O_3 exposure in summer for each threshold level

Threshold level	Log relative risk (95% CI)
10 ppb	4.5 (3.2, 5.8)
20 ppb	4.0 (2.6, 5.4)
30 ppb	3.2 (1.3, 5.2)

8.5.1.2 Scales used for presenting results derived from uncertainty analysis of estimates of changes in the health impacts attributed to future air quality

The four scales used to present estimated changes in the number of respiratory ED visits in Section 8.4 are also used in this section. As stated earlier, the scale of 'per head of population' allows evaluation of the other factors separate from the impact of population growth. Equation 8.11 is an example equation used to describe how to calculate this population adjusted metric for Groups B and C. This equation is a combination of Equations 8.5 and 8.7. In Equation 8.11, Pop_i and TP are specific for each decade (d). The same principle described here for Equation 8.11 under Groups B and C to calculate the population-adjusted estimate is also applied for Groups D, E and F. An additional scale, the population-adjusted percentage change in the number of respiratory ED visits per day was also included in the results presented in this section.

$$J_d = R\sum_{i=1}^{N}\left(\frac{\sum_{t=1}^{T(d)} e^{\beta C_{i,t}(d)} - 1}{T(d)}\right)\frac{P_i(d)}{K(d)} \qquad (0.11)$$

8.5.2 Results

8.5.2.1 Estimated respiratory morbidity associated with $PM_{2.5}$ and O_3 under Groups A to C

Figure 8.8 shows that, of the three drivers examined under Groups A, B and C, population growth had the most influence on changes in respiratory ED visits associated with exposure to the pollutants of concern between decades 1 and 2 (see Appendix E.6 for the estimated changes presented in tabular form). Under Group A, i.e., including only the impacts of climate change as predicted by the CSIRO model, there were relatively small increases in the number of respiratory ED visits per day of 0.08 (95% CI: 0.01, 0.16), 0.18 (95% CI: 0.01, 0.36) and 0.33 (95% CI: 0.22, 0.44) for $PM_{2.5}$ in summer, $PM_{2.5}$ in winter and O_3 in summer respectively. These increases per day corresponded to 8 (95% CI: 1, 15), 17 (95% CI: 1, 33) and 30 (95% CI: 20, 40) additional ED visits per season. It is clear that these estimated changes which resulted from climate change alone were minimal compared to those when population growth was also taken into account under Group B. For example, a 48.1% (95% CI: 48.1, 48.2) relative change in $PM_{2.5}$ -related respiratory ED visits in summer between the two decades was predicted under Group B (see Figure 8.9a). This change was considerably higher than the 3.1% (95% CI: 3.1, 3.1) increase estimated under Group A. The large difference in percentage change between these two groups was the 45% increase in population projected in the 2030 MLF population scenario.

Overall, changes in air pollution emissions had a greater influence on changes in respiratory ED visits associated with $PM_{2.5}$ and O_3, compared to the impact of climate change. This can be clearly seen from Figure 8.9 where the changes in respiratory ED visits are presented as population-adjusted percentage change.

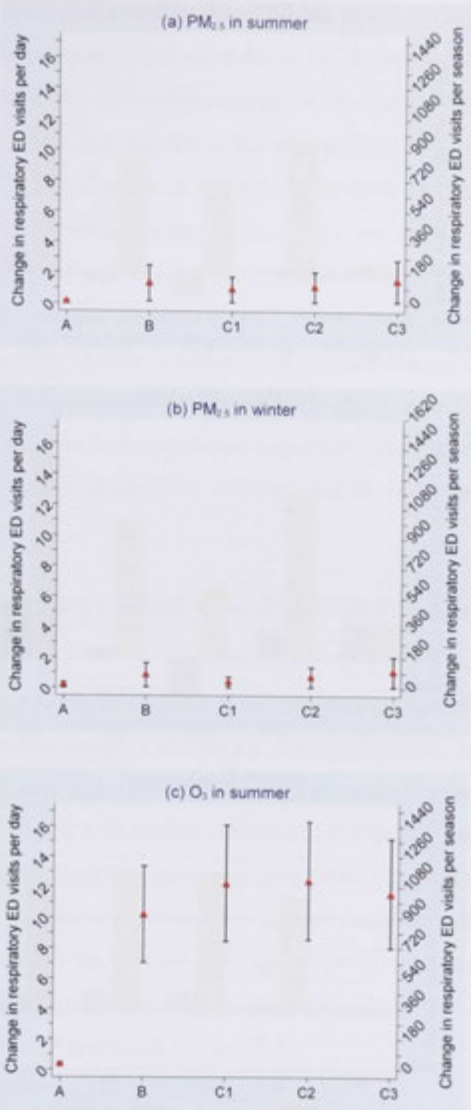
In summer, the CSIRO model predicted a reduction in respiratory ED visits associated with $PM_{2.5}$ under Groups C1 and C2 (see Figure 8.9a). The lowest air pollution emissions, predicted from the low impact emissions scenario of Group C1, resulted in the greatest reduction in the population-adjusted percentage change in respiratory ED visits associated with $PM_{2.5}$ exposure in the summer, of 7.6% (95% CI: -7.7, -7.5). The reduction corresponded to 0.050 (95% CI: -0.006, -0.096) fewer population-adjusted respiratory ED visits per day per million population, as presented in Table 8.10.

In winter, the changes in air pollution emissions had a more pronounced effect on health due to $PM_{2.5}$ exposure, compared to the health effects estimated in summer. Higher pollution emissions in winter in decade 2, as assumed under Group C3, resulted in a 20.1% (95% CI: 19.9, 20.2) increase in the population-adjusted respiratory ED visits per day due to $PM_{2.5}$ exposure (see Figure 8.9b). In contrast, if pollution was markedly reduced as assumed under Group C1, a 12.9% (95% CI: -12.8, -12.9) reduction in population-adjusted respiratory ED visits from decade 1 to decade 2 was predicted. Under Group C2 in the winter, the reduced air pollution emissions resulted in a decrease in $PM_{2.5}$-related respiratory ED visits, but the magnitude of the reduction was less than the increased health risk of $PM_{2.5}$ exposure induced by climate change. As a result, the estimate was of a 3.3% (95% CI: 3.3, 3.4) increase in $PM_{2.5}$-related respiratory ED visits (population-adjusted) between decade 1 and decade 2.

Table 0.10 Estimated changes* (95% CI) in the number of $PM_{2.5}$- and O_3-related respiratory ED visits per day (population-adjusted) between decades 1 and 2 predicted by the CSIRO model under Groups A to C.

Group	Estimated change (95% CI)		
	$PM_{2.5}$ Summer	$PM_{2.5}$ Winter	O_3 Summer
A	0.020 (0.002, 0.039)	0.045 (0.003, 0.087)	0.080 (0.054, 0.108)
B	0.015 (0.002, 0.029)	0.034 (0.003, 0.066)	0.105 (0.071, 0.142)
C1	-0.050 (-0.006, -0.096)	-0.045 (-0.003, -0.087)	0.447 (0.305, 0.597)
C2	-0.020 (-0.002, -0.038)	0.012 (0.001, 0.023)	0.478 (0.326, 0.640)
C3	0.049 (0.006, 0.094)	0.070 (0.005, 0.137)	0.336 (0.229, 0.450)

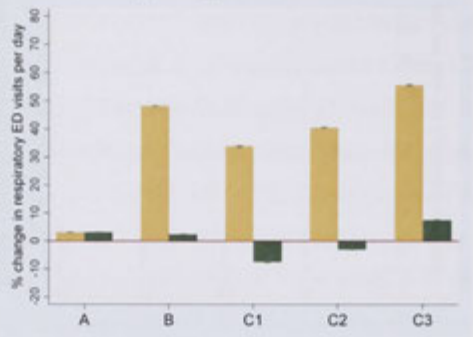
Note: *The changes are presented in the unit of per million population per day.



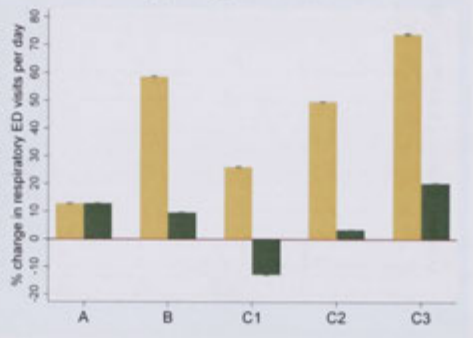
A – Climate change only
B – Climate change + 2030 MLF population growth
C1 – Climate change + 2030 MLF population growth + low impact emissions
C2 – Climate change + 2030 MLF population growth + MLF emissions
C3 – Climate change + 2030 high impact population growth + high impact emissions

Figure 0.8 Estimated changes (95% CI) in respiratory ED visits (unadjusted for population) per day and per season associated with exposure to $PM_{2.5}$ and O_3 between decades 1 and 2 predicted by the CSIRO model under Groups A to C

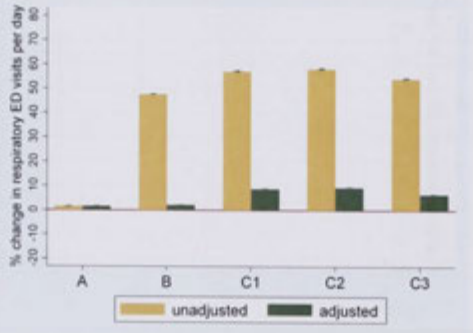
(a) $PM_{2.5}$ in summer



(b) $PM_{2.5}$ in winter



(c) O_3 in summer



A – Climate change only
B – Climate change + 2030 MLF population growth
C1 – Climate change + 2030 MLF population growth + low impact emissions
C2 – Climate change + 2030 MLF population growth + MLF emissions
C3 – Climate change + 2030 high impact population growth + high impact emissions

Figure 0.9 Percentage change (95% CI) in respiratory ED visits (unadjusted and adjusted for population) associated with exposure to $PM_{2.5}$ and O_3 between decades 1 and 2 predicted by the CSIRO model under Groups A to C

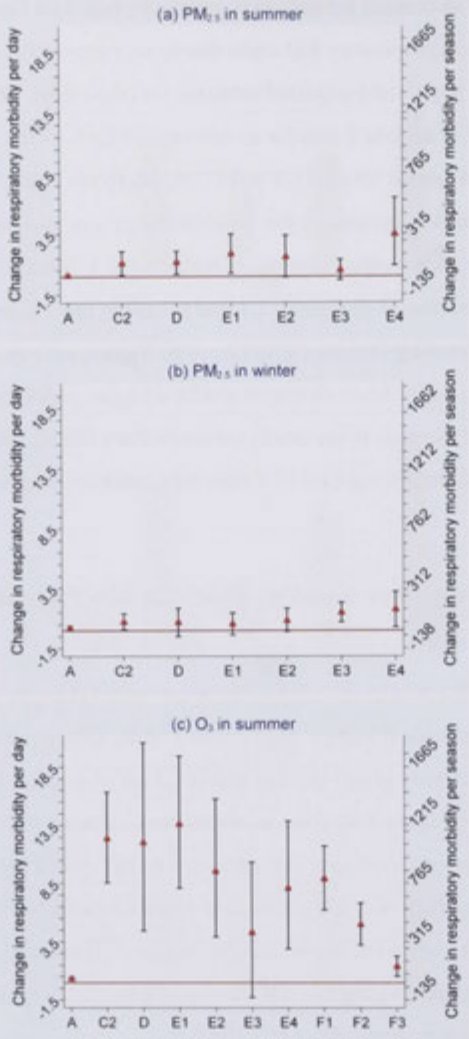
For O_3 in summer, the decreased air pollution emissions based on Groups C1 and C2 did not lead to a reduction in respiratory ED visits due to O_3 exposure as reported above for $PM_{2.5}$. In contrast, the population-adjusted estimate for respiratory ED visits in the summer related to O_3 exposure in decade 2 was for an increase of 8.6% (95% CI: 8.5, 8.8) and 9.2% (95% CI: 9.1, 9.4) under Groups C1 and C2 respectively, compared to the baseline (see Figure 8.9c). A similar increase in the health impacts was also predicted under Group C3, but was of a lower magnitude. As noted in Section 8.2.3, reduced NO_x emissions under the cleaner emissions scenarios in Groups C1 and C2 mean there is less titration of the background O_3 level, causing O_3 concentrations to be higher, mainly in the inner areas of the city. The higher O_3 concentrations together with a higher population density in the inner areas result in an increase in the predicted respiratory effect. Note that this increase is likely to be sensitive to the assumption of a zero concentration threshold for O_3-related health effects.

8.5.2.2 Estimated respiratory morbidity associated with $PM_{2.5}$ and O_3 under Groups D to F

The focus of results presented in this section is on estimated changes in respiratory morbidity outcomes between decade 1 and decade 2 under Groups D to F. However the estimates reported previously under Groups A and C2 are also presented here again for the purpose of comparison. Figure 8.10 contains estimates of changes between decades 1 and 2 in the number of respiratory morbidity outcomes associated with $PM_{2.5}$ and O_3 exposure per day and per season. Results of the population-adjusted estimated changes are shown in Table 8.11. Figure 8.11 shows the estimated changes as percentage change, including both unadjusted and adjusted for population. The results displayed in Figures 8.10 and 8.11 are presented in tabular form in Appendices E.7 and E.8.

- $PM_{2.5}$ in summer

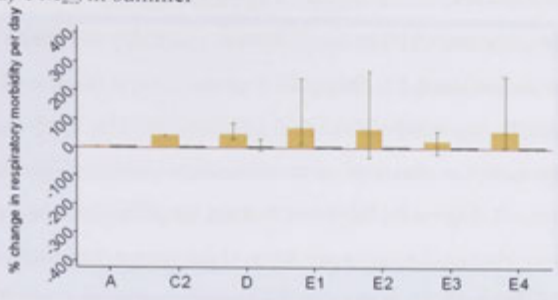
There was only a small difference in the estimates of the change in the number of respiratory morbidity associated with $PM_{2.5}$ exposure in summer per day and per season between decades 1 and 2 under Group C2 and Group D (see Figure 8.10a). That is, taking account of the temperature modifying effects assumed under Group D, resulted in only a minor additional variation to the estimated change in $PM_{2.5}$-related respiratory morbidity in summer, compared to the combined impact of climate change, population growth and air pollution emissions assumed under Group C2.



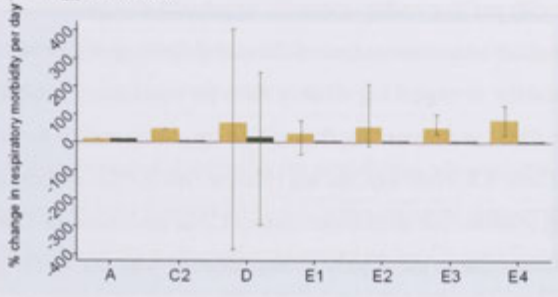
A – Climate change only
C2 – Climate change + 2030 MLF population growth + MLF emissions
D – Climate change + 2030 MLF population growth + MLF emissions + temperature modifying effect
E – Climate change + 2030 MLF population growth + MLF emissions + age distribution + choice of dose-response functions (Groups E1 to E4)
F – Climate change + 2030 MLF population growth + MLF emissions + O_3 threshold levels (F1=10 ppb, F2=20 ppb and F3=30 ppb)

Figure 0.10 Estimated changes (95% CI) in respiratory morbidity outcomes (unadjusted for population) per day and per season associated with exposure to $PM_{2.5}$ and O_3 between decades 1 and 2 as predicted by using the CSIRO model under Groups A, C2, D, E and F

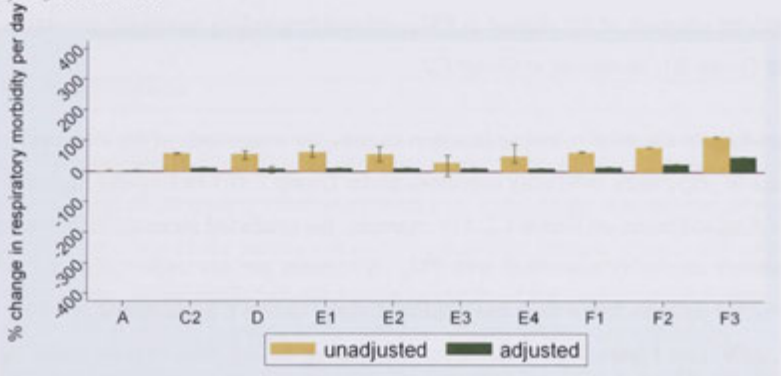
(a) $PM_{2.5}$ in summer



(b) $PM_{2.5}$ in winter



(c) O_3 in summer



A – Climate change only
C2 – Climate change + 2030 MLF population growth + MLF emissions
D – Climate change + 2030 MLF population growth + MLF emissions + temperature modifying effect
E – Climate change + 2030 MLF population growth + MLF emissions + age distribution + choice of dose-response functions (Groups E1 to E4)
F – Climate change + 2030 MLF population growth + MLF emissions + O_3 threshold levels (F1=10 ppb, F2=20 ppb and F3=30 ppb)

Figure 0.11 Percentage change (95% CI) in respiratory morbidity outcomes (unadjusted and adjusted for population) associated with exposure to $PM_{2.5}$ and O_3 between decades 1 and 2 predicted by the CSIRO model under Groups A, C2, D, E and F

Uncertainty in the estimates due to the future change in age structure can be observed by comparing the differences in estimated changes in respiratory morbidity between Groups C2 and E1. As described earlier in Section 8.5.1, Group E1 examined the influence of age distribution by using age-specific log relative risks that were estimated by applying the blending approach. This approach was also applied to estimate the uniform log relative risks for all-ages used under Group C2. Figure 8.10a shows that the magnitude of the best estimate of the change in respiratory morbidity outcomes per day and per season was higher under Group E1 than for Group C2. Figure 8.12 shows that the largest contribution of estimated change in respiratory morbidity per day under Group E1 expressed as a percentage was from the elderly group. This is a result of a combination of the population ageing predicted in the MLF population scenario and the strongest log relative risks for respiratory morbidity associated with exposure to $PM_{2.5}$ in summer for this age group, compared to the other two age groups, as reported in Table 8.8. Note that the log relative risk for the elderly was also greater than the uniform log relative risk used under Group C2 as presented in Table 8.1. The impact of the strong log relative risk in the elderly group, coupled with the 140% growth in the population of this group projected under the 2030 MLF population scenario, resulted in the greater estimate of the change in $PM_{2.5}$-related respiratory morbidity per day in summer under Group E1, compared to Group C2.

Depending on the dose-response function chosen, the magnitude of the estimates of the change in respiratory morbidity outcomes under Group E (E1 to E4) was higher or lower than that estimated based on Group C2. For example, the predicted increase in the number of respiratory morbidity associated with $PM_{2.5}$ in summer per day under Group E3 of 0.51 (95% CI:-0.27, 1.38) was lower than that predicted under Group C2, estimated at 1.09 (9.5%CI: 0.12, 2.08) (see Figure 8.10a). However when using the log relative risks under Group E4, the $PM_{2.5}$ effect resulted in an increase of 3.48 (95% CI: 0.92, 6.49) respiratory morbidity outcomes per day which was higher than the estimate under Group C2. In the scale of percentage change unadjusted for population as presented in Figure 8.11a, using the different dose-response functions coupled with the change in age structure resulted in a 50% variation between the lower (Group E3) and upper (Group E1) estimates of the changes in $PM_{2.5}$-related respiratory morbidity in summer.

To investigate underlying causes of the variation in the estimated changes under Group E, it is necessary to considering the log relative risks for each age group across the different dose-

response functions. Referring back to Table 8.8, it can be seen that the log relative risk for respiratory morbidity associated with $PM_{2.5}$ in the summer in the ≥65 year age group under Group E4 was stronger than that from the other sub-groups under Group E. In addition, within Group E4, the log relative risk for the elderly group was stronger than that for the other age groups in the population. Taken in combination, an increase in the size of the elderly population as predicted in the 2030 MLF population scenario and a stronger risk estimate in the elderly aged ≥65 years as assumed in Group E4 resulted in the largest estimated change in respiratory morbidity per day related to $PM_{2.5}$ exposure in summer (see Figure 8.10a). Yet, even with a large elderly population, the estimated change can be very small if a weak relative risk estimate is applied to estimate the health impact, as assumed under Group E3.

As expected, the estimated changes in the population-adjusted $PM_{2.5}$-related respiratory morbidity in summer for Groups E1 to E4 using different dose-response functions were little different from each other or from the estimates under Group C2 (see Figure 8.11a and Table 8.11). This was also true for the other pollutants and season (see Figures 8.11b,c and Table 8.11).

- $PM_{2.5}$ in winter

The pattern of the variation in the estimated changes in respiratory morbidity associated with $PM_{2.5}$ in the winter was different from that associated with $PM_{2.5}$ in the summer (as presented above). Here the magnitude of the point estimates of percentage change in the health impacts of $PM_{2.5}$ exposure was similar under Groups D and E. Under Group D, the estimate was of a 73.8% (95% CI: -383.9, 406.3) increase in the number of daily respiratory morbidity outcomes resulting from $PM_{2.5}$ exposure between the two decades. Such a percentage change was approximately 24 % higher than the estimates under Group C2 (see Figure 8.11b). Similarly, the largest estimated change in daily respiratory morbidity due to $PM_{2.5}$ exposure in winter under Group E4 was estimated at 82.2% (95% CI: 29.9, 134.0)—around 32% higher than the estimate under Group C2 (see Figure 8.11b).

Table 0.11 Estimated changes* (95% CI) in the number of respiratory morbidity outcomes per day, adjusted for population, associated with exposure to $PM_{2.5}$ and O_3 between decades 1 and 2 predicted by the CSIRO model under Groups A, C2, D, E and F.

Group	Estimated change (95% CI)		
	$PM_{2.5}$ Summer	$PM_{2.5}$ Winter	O_3 Summer
A	0.020 (0.002, 0.039)	0.045 (0.003, 0.087)	0.080 (0.054, 0.108)
C2	-0.020 (-0.002, -0.038)	0.012 (0.001, 0.023)	0.478 (0.326, 0.640)
D	-0.004 (-0.059, 0.055)	0.045 (-0.080, 0.170)	0.418 (-0.136, 1.060)
E1	-0.120 (-0.214, -0.029)	0.074 (0.018, 0.131)	2.187 (1.400, 3.080)
E2	-0.010 (-0.019, 0.000)	0.007 (0.001, 0.013)	0.153 (0.075, 0.243)
E3	-0.010 (-0.017, -0.003)	0.011 (0.004, 0.018)	0.164 (0.078, 0.247)
E4	-0.025 (-0.050, -0.003)	0.007 (0.001, 0.013)	0.178 (0.074, 0.315)
F1	NA	NA	0.046 (0.032, 0.060)
F2	NA	NA	0.037 (0.024, 0.051)
F3	NA	NA	0.014 (0.006, 0.023)

Note: *The changes are presented in the unit of per million population per day.

The magnitude of the point estimates for increased respiratory morbidity related to $PM_{2.5}$ exposure in the winter under Group D, which included the temperature modifying effect, was consistent with that presented earlier in Section 8.4.2. In Section 8.4.2, the percentage change was highest for $PM_{2.5}$ in the winter, compared to $PM_{2.5}$ and O_3 in the summer. Here, similarly, the largest change, in the population-adjusted respiratory morbidity outcomes between decades 1 and 2 was for $PM_{2.5}$ in winter (20.1% (95% CI: -296.2, 250.0)) under Group D, compared to those for $PM_{2.5}$ (-0.5% (95% CI:-10.9, 29.1)) and O_3 (8.1% (95% CI: -4.4, 14.4)) in the summer.

To examine how the change in age distribution in 2030 could impact on the predictions of the respiratory morbidity associated with $PM_{2.5}$ in winter, the estimated change for Group C2 was compared with the estimate for Group E1. Under Group E1, the estimate of change in $PM_{2.5}$-

related respiratory morbidity outcomes per day and per season in the winter, as shown in Figure 8.10b, was slightly smaller than the estimate for Group C2. Despite a considerable increase in the elderly population predicted by the 2030 MLF population scenario, the inclusion of age distribution in Group E1 resulted in a smaller increase in respiratory morbidity, compared to the estimated increase using the uniform age distribution as assumed under Group C2. This was mainly due to the weaker log relative risk associated with $PM_{2.5}$ exposure in winter estimated for the elderly group, compared to that estimated for the children aged below 14 years (see Table 8.8). Likewise, the log relative risk estimated for the elderly population used for Group E1 was slightly lower than the uniform log relative risk for all-ages used under Group C2. Figure 8.12 shows that the magnitude of the increased estimate associated with $PM_{2.5}$ exposure in the *winter* for the elderly group was appreciably lower than the estimate attributable to $PM_{2.5}$ exposure in the *summer* under Group E1.

Using different dose-response functions under Group E resulted in variation in the estimates of changes in respiratory morbidity related to $PM_{2.5}$ between the two decades in the winter, ranging from a 32.8% (95% CI: -42.4, 80.6) increase under Group E1 to an 82.2% (95% CI: 29.9, 134.0) increase under Group E4 (see Figure 8.11b). The difference between the largest and smallest percentage change of 50% was similar to that estimated for $PM_{2.5}$ in summer. An increase of 1.74 (95% CI: 0.28, 3.22) respiratory morbidity outcomes per day in winter related to $PM_{2.5}$ exposure (see Figure 8.10b) estimated under Group E4 (the largest change among the sub-groups under Group E) was attributable to the stronger log relative risk estimated in the elderly group taken from the study of EPA Victoria, compared to the log relative risks for this age group used for Groups E1, E2 and E3.

- O_3 in summer

The results shown in Figures 8.10c and 8.11c suggest that, among the factors analysed, altering O_3 threshold levels under Group F had the largest effect on the estimates of changes in respiratory morbidity due to O_3 exposure in the summer. With a threshold of 30 ppb, the predicted increase was for an additional 1.38 (95% CI: 0.54, 2.24) respiratory morbidity outcomes per day in decade 2 compared to decade 1. This was the smallest estimated change under any of the other groups, except for those calculated based on Group A that considered only the impact of climate change (see Figure 8.10c). However, when this increase is expressed as percentage change, it has the greatest magnitude of all of the groups (see Figure 8.11c). To explain this apparent paradox between estimates expressed as difference in the number of respiratory morbidity per day versus percentage change in the number of respiratory morbidity per day, a further investigation was performed as shown in Table 8.12. The actual estimates of respiratory morbidity outcomes per day in each decade played a role. For example, due to the small number of respiratory morbidity per day in decade 1 for the 30 ppb threshold (point estimate of 1.20 respiratory morbidity outcomes per day), even a small change of 1.38 additional morbidity outcomes per day between the two decades resulted in a large percentage change of 115.5%. In contrast, for the 0 ppb threshold scenario, there was a higher initial number of respiratory morbidity outcomes per day (point estimate of 21.31 respiratory morbidity outcomes per day) in decade 1. Despite a larger increase in respiratory morbidity outcomes from decade 1 to decade 2 (12.36 additional morbidity outcomes per day), this translated into only a 58.0 % relative change. Of the three O_3 thresholds assessed here, the lowest predicted difference in O_3-related respiratory morbidity outcomes between decades 1 and 2 was for the highest O_3 threshold of 30 ppb. The results suggest that the higher the level of O_3 threshold applied, the lower the absolute difference in the number of O_3-related health burden between the future and the baseline periods estimated.

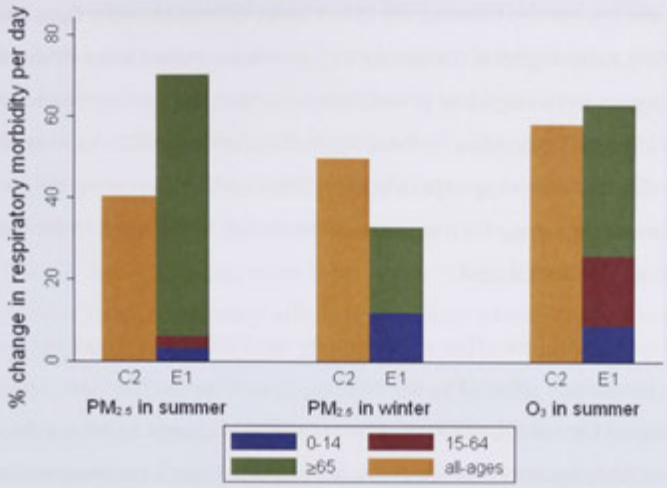


Figure 0.12 Percentage change in respiratory morbidity associated with $PM_{2.5}$ and O_3 exposure between decades 1 and 2 predicted under Groups C2 (all-ages) and E1 (segregated by three age groups)

An estimated change in respiratory morbidity due to the O_3 effect under Group D was slightly lower than that estimated under Group C2. This result was consistent with the results presented in Section 8.4.2 in that taking account of the temperature modifying effect lowered the estimated increase in respiratory mobility between the two decades, compared to with no consideration of the effect modification.

Table 0.12 Best point estimates of the number of respiratory morbidity outcomes per day in decades 1 and 2 as well as the changes between decades 1 and 2 for the different O_3 thresholds under Group F

Threshold (ppb)	Estimate in decade 1	Estimate in decade 2	Additional respiratory morbidity per day between decades 1 and 2	Percentage change in respiratory morbidity per day between decades 1 and 2
0	21.31	33.67	12.36	58.0
10	13.86	22.77	8.91	64.2
20	6.17	11.15	4.98	80.8
30	1.20	2.58	1.38	115.5

The estimate of change in respiratory morbidity associated with O_3 exposure in summer between decades 1 and 2 under Group C2 was compared with the estimate under Group E1 to explore the effect of the predicted future change in age distribution. It can be seen from Figure 8.12 that there was only a small difference in the percentage change in O_3-related

respiratory morbidity per day between the two decades estimated under these two groups. As shown in Table 8.8, the log relative risks for O_3 in summer under Group E1 for the three different age groups were very close to each other. Further, the age-specific log relative risks applied under Group E1 were close to those applied under Group C2. As a result, the analysis including age distribution and age-specific log relative risks under Group E1 provided similar estimates to those from Group C2 that assumed no change in the age distribution and using a single log relative risk for all ages.

However, the estimates of the effect on respiratory morbidity of exposure to O_3 in the summer were moderately affected by the different dose-response functions under Group E (E1 to E4). Among Groups E1 to E4, the lowest estimated change in the number of respiratory morbidity outcomes per day between decades 1 and 2 occurred under Group E3: 4.29 (95% CI:-1.28, 10.32) additional morbidity outcomes (see Figure 8.10c), corresponding to a 28.7% (95% CI: -15.6, 52.8) (see Figure 8.11c) increase. This percentage change was 34% lower than that predicted for Group E1 (62.9% with 95% CI: 45.7, 82.2) which was the largest percentage change compared to the other sub-groups within Group E. The variation in the estimated change in the number of respiratory morbidity outcomes per day, as well as in percentage change, under Group E was determined by the magnitude of the relative risks for the $\geq$65 years age group. The largest increase in the number of respiratory morbidity outcomes per day of 13.57 (95% CI: 8.09, 19.43) for Group E1 was driven by the stronger log relative risk for O_3-related respiratory morbidity (point estimate of $4.4*10^{-3}$), compared to the other log relative risks for the elderly age group under Groups E2 to E4 (see Table 8.8). In contrast, the weak relative risk of $0.5*10^{-3}$ (point estimate) in the elderly group used under Group E3 resulted in the lowest estimated change in respiratory morbidity noted above.

8.6 Discussion

8.6.1 The impact of climate change-induced changes in air pollutant concentrations on the acute health effects of air pollution

By applying a conventional method which considered only changes in air pollution concentrations induced by climate change, the number of $PM_{2.5}$-and O_3-related respiratory ED visits in summer in the PPR in the 2070s compared to the 2000s was estimated to increase under the assumptions of the A2 SRES GHG emissions scenario, unchanged anthropogenic air pollution emissions and constant population. Although the direction of the

estimated effect on respiratory morbidity related to $PM_{2.5}$ and O_3 in the summer based on the four GCMs employed was consistent, the magnitude varied. In relative terms between the two periods, the point estimates for summer predicted by the four GCMs ranged from a 2.5% to an 8.9% increase in respiratory ED visits related to $PM_{2.5}$ and a 3.2% to a 5.8% increase related to O_3. Three of the four GCMs used, predicted a decline in winter respiratory ED visits attributable to $PM_{2.5}$. The GFDL model was the exception, predicting an increase in respiratory ED visits due to $PM_{2.5}$ exposure in the winter. The magnitude of the estimated changes in the $PM_{2.5}$-related respiratory effect for the winter was generally lower than that predicted for the summer.

The extent of the estimated changes in $PM_{2.5}$- and O_3-related respiratory ED visits due to the impact of climate change according to two different methods—with and without taking account of the temperature modifying effect—applied in this chapter varied by season and the GCMs used. Two factors were identified that determined the differences in the estimated changes in the respiratory endpoint resulting from these two methods: the pattern of relative risks across the temperature range; and the magnitude and pattern of changes in the number of days for each temperature stratum between the baseline and the projected period, as predicted by the GCMs.

Similar to previous studies predicting short-term health impacts associated with O_3 exposure under a changing climate, the results presented in this chapter suggest that detrimental consequences for human health are likely. A similar study in Australia estimated around a 200% increase in respiratory hospital admissions in warm months per year due to exposure to O_3 max 8-h above 60 ppb in the Sydney Metropolitan Region in the 2060s relative to the 2000s (Cope et al. 2008b). Despite a similar set of assumptions and scenarios applied here, as well as similar methods in projecting O_3 concentrations, the estimate predicted by that study is noticeably greater than the largest estimate here of a 5.8% increase in O_3-related respiratory ED visits predicted by the GFDL model in the 2070s relative to the 2000s. This might be partly related to the application of different O_3 thresholds. As demonstrated in Section 8.5.2.2 on the analysis of uncertainty associated with O_3 thresholds, when a higher level of O_3 threshold is assumed, a larger percentage change is estimated. This highlights the importance of analysing uncertainties to investigate the sensitivity of the estimates to the application of different O_3 thresholds to quantify the impact of climate change on O_3-related health effects.

The results of increased respiratory ED visits related to O_3 exposure based on the conventional method (with no account taken of the temperature modifying effect) as presented in this chapter are generally in agreement with overseas studies predicting O_3-related respiratory morbidity at a regional level. For example, Sheffield et al. (2011) estimated O_3-related childhood asthma ED visits in New York City to increase at 7.3% (varied among 14 counties examined from 5.2% to 10.2%) in summer in the 2020s compared to the 1990s baseline, as a result of climate change. The prediction reported by that study was made based on the A2 SRES GHG emissions scenario, as was also employed here. Another study projecting health impacts attributable to O_3 max 1-h resulting from climate change, reported an average increase of 0.8% and 2.1% in respiratory and asthma hospital admissions respectively in 50 US cities between the 2050s and the 1990s (Bell et al. 2007a). Such an increase was due to a rise in O_3 concentrations at about 4.8 ppb based on the assumption of GHG emissions under the A2 SRES scenario, with population between the two periods held constant.

The absolute changes in the number of respiratory ED visits attributable to $PM_{2.5}$ presented here, despite being of lower magnitude than those related to O_3 exposure, suggest future potential health impacts of changes in $PM_{2.5}$ induced by climate change. Based on the conventional method (taking no account of the temperature modifying effect), the estimated changes between decades 1 and 3 suggest that there will be an increase in $PM_{2.5}$-related respiratory ED visits in the summer. The increased health risks associated with $PM_{2.5}$ exposure in summer may be offset to some degree by reduced respiratory morbidity as a result of lower $PM_{2.5}$ concentrations in the winter, particularly in the densely-populated suburbs of Melbourne.

When the temperature modifying effect is taken into consideration, the results here for summer indicate that the magnitude of the increased risks of $PM_{2.5}$-related respiratory ED visits, in the 2070s compared with the baseline period, may be greater than the effects of O_3. A larger increase in the health impacts associated with $PM_{2.5}$, compared to O_3 under a changing climate, has also been previously reported. Tagaris et al.(2009) estimated that changes in $PM_{2.5}$-related premature mortality between the 2050s and the 2000s were 15 times larger than those associated with O_3 in the US under the assumptions of the A1B SRES GHG emissions scenario, constant population and fixed anthropogenic air pollution emissions. The larger change in $PM_{2.5}$-related health impacts in that study, compared to O_3, was due to the

higher annual $PM_{2.5}$ concentrations in the future period in the majority of the states (2/3) relative to the baseline (Tagaris et al. 2007). In contrast, O_3 concentrations in the future under climate change were estimated to increase in a smaller number of the states and be limited to only summer time.

The difference in the estimates from the two methods used in this chapter to quantify changes in $PM_{2.5}$- and O_3-related respiratory ED visits for the 2070s compared with the baseline 2000s highlights the importance of considering temperature modifying effects when estimating the health impacts of air pollution and climate change. The reduction in the magnitude of estimated changes in O_3-related respiratory ED visits when taking into account the temperature modifying effect may reflect a role that behavioural changes and adaptation can play to avoid adverse heat and air pollution-related health effects under a changing climate. As explained in Chapter 6, the lower relative risk associated with O_3 exposure when temperature was high in the summer may be because people would prefer to spend more time indoors to avoid heat exposure. This may result in lower exposure to O_3 due to its low concentrations inside buildings. This behavioural change could possibly avert the health risks associated with heat and O_3 exposure simultaneously under a warmer world. Conversely, the increase in magnitude of $PM_{2.5}$-related respiratory illness in summer when the temperature modifying effect was included in the predictions, compared to the conventional method, indicates that there is a possibility of the persistence of the $PM_{2.5}$ risks on hot days in summer even with behavioural changes and adaption. Perhaps the health risks from $PM_{2.5}$ exposure in summer, compared to those for O_3, should be a priority that will require both air quality management and public health interventions to decrease risks. Some interventions at the individual level as discussed at the international workshop entitled "From Good Intentions to Proven Interventions: Effectiveness of Actions to Reduce the Health Impacts of Air Pollution", may have potential in this regard such as the use of air conditioning and coincident closing of windows to reduce infiltration of PM and O_3 by reducing air exchange (Giles et al. 2011). To prevent an increase in adverse health effects related to PM exposure due to climate change from an air quality management perspective, this may be undertaken through urban planning to separate residential areas from major roads, leading to the reduction of the level of indoor $PM_{2.5}$ which has its source from motor vehicles.

Behavioural changes to cope with heat exposure, for example using air-conditioning, may provide a co-benefit in reducing health risks of exposure to O_3. However relying on air-

conditioning to cope with heat will contribute to production of GHGs due to increased use of energy. As long as use of fossil fuels remains the main source of electricity generation, using air conditioning to cope with heat creates a positive feedback, with elevated emissions of GHGs leading to a warmer climate and associated health consequences.

8.6.2 Uncertainty analysis

The results of the uncertainly analysis presented in this chapter suggest that estimations of health impacts associated with air pollution under a changing climate are sensitive to non-climate factors and projection methods. Based on the analyses here, estimated changes in the health impacts of air pollution due to changes in future climate alone, driven by increased GHG emissions in the next 20 years were modest in comparison with the other potential factors analysed including population growth, air pollution emissions, the temperature modifying effect, a combination of changes in age structure and the choice of dose-response functions, and the application of O_3 thresholds. Note that the analyses in this chapter were conducted to explore uncertainties associated with the non-climate factors in the 2030s. If the analyses were undertaken to investigate uncertainties in the estimates for the 2070s, the relative importance of the factors considered here may be different, including the impact of climate change alone.

Overall, it was apparent that an expected large increase in future population in the PPR was the most important driver of changes in the health impacts of air pollution. Future changes in air pollution emissions, although of lesser importance compared to population growth, had a vital role to play in influencing the direction and magnitude of predicted future health impacts of air pollution. In summer, it was evident that the combined impact of an ageing population and the choice of dose-response functions introduced large changes to the estimates of $PM_{2.5}$- and O_3-related respiratory morbidity. These changes in the estimations were greater than those found in comparing the models with and without the temperature modifying effect. In winter, however, the influence of the different methods regarding the temperature modifying effect was greater than the combined factors of an ageing population and the choice of dose-response functions on the estimated changes in $PM_{2.5}$-related respiratory ED visits. For predicting changes in the respiratory effect attributable to O_3, the largest contributor to the variation in the estimations was from the application of different O_3 threshold levels.

Population growth has been identified in previous studies as a main source of uncertainty in estimations of the future health impacts of climate change. Results from a study by Knowlton et al. (2004) suggested that increased population was the largest source of uncertainty for the predictions of O_3-related mortality in summer in the 2050s under a changing climate, compared to the application of O_3 thresholds and changes in anthropogenic O_3 precursor emissions. However this is contradictory to the results of a recent study which compared contributions from three factors to the variation in predicted future changes in O_3-related mortality due to climate change across the US (Post et al. 2012). Analysis of variance (ANOVA) results from that study indicated that population changes projected for 2050 provided the smallest source of variation compared to the use of different climate-air quality modelling systems and the choice of dose-response functions.

From a population point of view, it is not only changes in population size that causes substantial uncertainties to the projection of future health impacts of air pollution related to climate change, but also changes in spatial distribution of the population. As presented in Section 8.5.2.1, the higher population density in inner urban areas as assumed in the 2030 high impact population scenario, along with increased air pollution emissions strongly influences the magnitude and direction of relative changes in respiratory morbidity associated with $PM_{2.5}$ and O_3 exposure between the 2030s and the baseline. The impact of the population distribution was also observed in a study quantifying the effects of climate change on premature mortality attributable to O_3 between 2050 and 2000 at a national scale in the US (Post et al. 2012). The authors of that study noted that the variation in estimated O_3-related mortality in three different regions was due largely to the non-uniform population distribution projected for 2050. Of the three regions, the West of the continent, where projections were for a smaller population in 2050, had the smallest increase in mortality attributable to O_3.

The results reported in this chapter that evaluated uncertainty due to changes in the age structure of future populations in the PPR suggested that this factor was likely to be an important potential source of variation in predicting future health impacts of air pollution. However the magnitude of the variation was largely conditional on the size of relative risk estimated for each age group for the health endpoint of interest. The analyses here show that there was a large impact on the health risk estimations only if a relative risk estimate of the health endpoint examined was relatively stronger in the age group predicted to have the largest change in population size. Further, the magnitude of the impact of an ageing

population depended upon the extent to which the relative risk estimated in the elderly population was larger than that estimated for all-ages.

Variation in the estimates caused by the changing age structure may be highly dependent on the health outcome under consideration for the same air pollutant. For instance based on the log relative risk estimate specific to each age group derived from the EPHC study under Group E3, as reported earlier, a change in respiratory morbidity associated with $PM_{2.5}$ exposure in summer between the 2030s and the baseline was considerably lower than that based on Group C2 assuming a uniform age structure (see Figures 8.10 and 8.11). The lower estimate was due largely to the use of the weak log relative risk for this morbidity outcome estimated for the elderly group (point estimate of $1.9*10^{-3}$) derived from the EPHC study. However based on the EPHC study, the log relative risk for cardiovascular morbidity associated with $PM_{2.5}$ in summer was estimated at $4.8*10^{-3}$ (95% CI: $0.8*10^{-3}$, $9.0*10^{-3}$) for the elderly group (Environmental Protection and Heritage Council 2010). That is the size of the increased risk for cardiovascular morbidity was more than double that estimated for respiratory morbidity. If cardiovascular morbidity was the health endpoint of interest in this study, there would be a larger estimated change in health impact of exposure to $PM_{2.5}$ in summer in models accounting for the ageing population than in those using a constant age structure.

The choice of dose-response functions is a potential source of uncertainty in the quantification of the health impacts of air pollution due to climate change. In most cases based on the analyses here, the magnitude of uncertainty resulting from the combination of changes in age structure and the choice of dose-response functions was of considerable importance, with only population growth more influential. This has also been evident in three recent studies undertaking an uncertainty analysis to evaluate the impact of the application of different relative risks on changes in health impacts due to climate change. Kolstad and Johansson (2011) found that uncertainty associated with the chosen relative risks was larger than that associated with inter-model variability when projecting the effect of climate change on diarrhoeal admissions/reported cases. Results of a study by Wu et al. (2013) suggested that the use of different values of the relative risk of mortality attributable to heat had the largest impact on the variability in estimating excess mortality related to heat waves in 2050s under climate change. The effect of choice of relative risk values on the uncertainty in that study was larger than the other two factors compared including GHG emissions scenarios and heat

wave definitions. Similarly, results of a study by Post et al. (2012) indicated that uncertainty associated with the choice of epidemiological studies used to estimate O_3-related mortality under a changing climate was larger than that associated with population growth and distribution projected in the future.

It has been evident in a number of studies, including the results presented in the analyses here, that reduced anthropogenic air pollution emissions could offset the increased health impacts resulting from elevated air pollution concentrations induced by climate change (Tagaris et al. 2010; Selin et al. 2009; West et al. 2007). Although a reduction in air pollution emissions as assumed in the 2030 MLF and the low impact emissions scenarios could lead to an increase in population exposure to O_3 in the PPR, overall implementation of emissions controls is likely to bring health benefits to the residents of the PPR, due to reductions in exposure to the other pollutants.

8.6.3 Strengths and limitations

In terms of methods used, the greatest strength of this analysis was to apply the findings of the temperature modifying effect to assess the health impacts of air pollution resulting from climate change. This method allowed an additional dimension of the assessment which could be increasingly important in a warmer world. As stated earlier, taking account of the temperature modifying effect can to some extent be used to evaluate the influence of behavioural changes and adaptation in reducing health threats induced by simultaneous heat and air pollution exposure on hot days.

Given the limited number of studies up to the present attempting to predict the impact of climate change on the $PM_{2.5}$ effect on health, the results presented here provide additional evidence to show that not only changes in O_3 responding to climate change should be of concern in public health but also $PM_{2.5}$.

The way in which the uncertainty assessment in this chapter was designed and conducted was another important strength. Uncertainties at each stage of the prediction of the health impacts of air pollution under a changing climate were addressed systematically. At the stage of climate projections, an ensemble of four GCMs was employed to assess uncertainties inherent in climate modelling. At the stage of air quality projections, different assumptions of air pollution emissions were explored to gauge the extent to which various future trends in air

pollution emissions could alter the estimated changes in the health effects. At the stage of health impact projections, the main drivers identified from literature were systematically investigated.

Undertaking the uncertainty assessment not only provides benefits in reporting a full range of variations in the potential impact of climate change on health, but can give useful information to guide policy formulation in relevant sectors and to develop public health interventions. These implications will be discussed in the next chapter.

Although it is well established that exposure to air pollution causes numerous effects on health, the results presented here focused on a single acute health outcome which was respiratory morbidity. An estimation of the full array of health effects, short-term and long-term, potentially affected by the future climate change, is extremely challenging.

Due to limited computational resources in modelling air quality under climate change, there were some uncertainties which were not considered here such as those associated with different scenarios of GHG emissions. The scenario of GHG emissions assumed in the analyses here was conservative to represent the middle-range of future GHG emissions. In addition, the IPCC has developed a new set of GHG emissions scenarios; applying these to the estimation of changes in health impacts due to air pollution may provide different results compared to those presented here (IPCC 2013). Likewise the uncertainty assessment due to changes in future populations was not fully analysed. In the analyses of this chapter, there were only three scenarios of changes in population growth and spatial distribution of population assumed, population constant at 2006, the 2030 MLF population growth and the 2030 high impact future population scenario. Additional uncertainties include the possible change in the dose-response functions over time, rather than the single dose-response function used here. Dose-response functions may change over time due to adaptation through behavioural changes and building characteristics. Further discussion on the policy implication of this issue is provided in the next chapter, Section 9.4.2.2.

To explore the sensitivity of estimated changes in respiratory morbidity between decades 1 and 2 to the choice of dose-response functions, relative risks for both respiratory hospital admissions and for respiratory ED visits were used. Ideally, all of the the relative risks used to test this factor for uncertainty should be for the same type of morbidity outcome, i.e., *ED*

visits for this thesis. This is one of the limitations for the analyses conducted in this chapter. As noted in Section 8.5, the use of different types of hospital data here was pursued based on the assumption that relative risks for these two different hospital types for respiratory disease, particularly for the elderly, were similar, resulting in little impact on the estimated changes. The predictions undertaken in this chapter focused on the changes in respiratory morbidity. If other morbidity outcomes were of interest, the use of relative risks for the mixture of morbidity outcomes to evaluate uncertainty associated with the choice of dose-response functions may be inappropriate.

8.7 Chapter summary

This chapter estimated changes in respiratory ED visits attributable to changes in air pollution concentrations driven by climate change in the PPR between 1996 to 2005 and 2065 to 2074. To isolate the impact of climate change, one set of analyses assumed unchanged anthropogenic air pollution emissions and constant population over the two time windows selected. Using air pollution concentrations predicted by an ensemble of four GCMs, there was a consensus of an increase in respiratory ED visits associated with O_3 and $PM_{2.5}$ exposure in summer. The magnitude of the additional respiratory ED visits associated with O_3 exposure in summer was approximately five times larger than those associated with $PM_{2.5}$. In winter, a reduction of $PM_{2.5}$-related respiratory ED visits between the 2000s and 2070s was estimated based on predictions derived from three GCMs. Overall the magnitude of the estimated reduction in $PM_{2.5}$-related respiratory morbidity in winter was lower than the estimates of increases associated with $PM_{2.5}$ in summer. The predicted increase in the health risks of $PM_{2.5}$ and O_3 in summer was largely explained by the warmer climate, driving increases in concentrations of these two air pollutants. Conversely, the higher temperatures in winter predicted in the 2070s resulted in reductions in $PM_{2.5}$ concentrations in winter and consequently diminished the health risks of $PM_{2.5}$ such that estimates of respiratory morbidity were lower in the 2070s compared to the 2000s.

Estimates of changes in respiratory ED visits between the 2000s and the 2070s with inclusion of the temperature modifying effect were different from those estimated by the conventional method applying uniform relative risks across the temperature range. By using three relative risks estimated for $PM_{2.5}$ in summer for three temperature strata, there was an increase in estimated respiratory ED visits in the 2070s in relative to the 2000s. This increase was of greater magnitude than that predicted by using the conventional method. There was a small

change in O_3-related health risks in summer predicted by the method including the temperature modifying effect between the two decades. The estimates of changes in $PM_{2.5}$-related respiratory ED visits in winter made by using the non-uniform relative risks across the temperatures were paradoxical to those estimated by the conventional method. Two factors were identified to influence the extent of the differences of the estimates made between the two different methods. The first factor was the pattern of relative risks for each pollutant across the temperature range for each season and the other was the magnitude and pattern of changes in the number of days in each temperature stratum between the two decades.

This chapter also analysed variations contributed from non-climate factors that had potential to influence the estimations of changes in $PM_{2.5}$- and O_3-related respiratory morbidity between 1996 to 2005 and 2025 to 2034. Compared to other factors, the estimated changes in respiratory morbidity induced by changed air pollution concentrations driven by climate change in the 2030s relative to the 2000s were the smallest. Population growth predicted in the 2030s was generally identified to be the largest source of variation in the estimates of changes in respiratory illness associated with air pollution. For the estimated changes in O_3-related respiratory morbidity in summer, the application of different O_3 thresholds resulted in variation that was potentially as large as that due to population growth. The magnitude of the changes in the respiratory morbidity associated with $PM_{2.5}$ and O_3 between the two decades predicted as a result of changes in air pollution emissions assumed in the 2030s was greater than that resulting from the changing climate. This indicates that reductions in future air pollution emissions could be one potential measure to avoid the adverse health risks of air pollution due to climate change. The estimated changes in $PM_{2.5}$- and O_3-related respiratory morbidity between decades 1 and 2 were sensitive to the change in age structure projected in decade 2 and the choice of dose-response functions. The effect of an ageing population projected in the 2030s was prominent only if the relative risk for the health endpoint of interest in the elderly used to estimate changes in the health impacts of air pollution was strong and it had to be stronger than the relative risk estimated for all-ages

Chapter 9 Conclusions

9.1 Introduction

This thesis was aimed at assessing changes in the acute health effects of exposure to $PM_{2.5}$ and O_3 under a changing climate in the Melbourne Region. The research aim was established to address key research needs identified in the literature as outlined in Chapter 1. Addressing these needs is important to advance knowledge in the area of impact assessment of climate change on air pollution-related health outcomes, especially at an urban scale. The research needs identified in Chapter 1 are summarised as follows:

i) improving accuracy of the baseline dose-response relationships between air pollution and health, through applying estimates of air pollution exposure simulated by an air quality modelling system, used further for projections of future health risks of air pollution induced by climate change,

ii) applying the growing knowledge of the role of temperature as an effect modifier of the associations between air pollution and health to health impact assessment of climate change,

iii) estimating the impact of climate change on adverse health effects attributable to air pollution, particularly $PM_{2.5}$ and O_3, at an urban scale, and

iv) assessing uncertainties in projections of the health effects of air pollution under a changing climate.

In the context of estimating the health impacts of air pollution due to climate change in the Melbourne Region, four objectives were set to attain the research aim and to address the research needs. Previous chapters have demonstrated and presented methods and results of the analyses to address the research aim and objectives. The following section in this last chapter synthesises key findings presented in Chapters 5, 6 and 8. Next, strengths and limitations of this thesis that have been presented and discussed in the preceding chapters are summarised. Some applications of the research findings in practice and possible policy implications are outlined, and areas for future research are identified. This chapter concludes with some remarks about the significance and original contribution of this thesis.

9.2 Synthesis of key findings

The synthesised findings of this thesis are presented with respect to each research objective as follows.

Objective 1: Selecting the best approach to estimate the relationships between air pollution and health

A blending approach, in which air pollution and weather data are generated by merging data from simulations and observations and where the analyses are conducted at the SLA level, was chosen. This approach was felt to more accurately represent exposure as it provided larger relative risk estimates, compared to other three approaches, for cardiovascular and respiratory mortality and ED visits associated with exposure to $PM_{2.5}$ and O_3. The larger size of the relative risks estimated by the blending approach was thought to signify lower measurement error in the exposure measurement as it was able to take account of intra-urban variability of air pollution exposure. It is well established that the presence of exposure error can bias the associations between air pollution and health towards the null. The larger size of relative risks estimated by using the blending approach may reflect the ability of this approach to reduce the measurement error.

Objective 2: Investigating whether temperature modifies the effects of air pollution on health outcomes

Relative risk estimates for health outcomes associated with exposure to $PM_{2.5}$ and O_3 in the Melbourne Region were non-uniform across the temperature range. In other words, the results suggest that temperature modifies the effects of air pollution on health. The temperature modifying effect appeared to be limited to only the ED visits outcomes, predominantly respiratory ED visits. In the summer, the observed pattern in relative risks for respiratory ED visits in different temperatures is that the relative risk is stronger along with increased temperature. This is in contrast to the pattern found in the winter where the relative risk is greater at lower temperatures.

Biologically, the stronger association between air pollution and health at both the low and high extremes of temperature as found in this thesis is plausible. These findings may be explained by these two exposures affecting health via the same patho-physiological pathway, mainly through the sympathetic nervous system. This shared pathway may lead

to synergistic effects when the human body is simultaneously exposed to these two environmental factors.

There is one unexpected finding in which the strongest relative risk for respiratory ED visits associated with O_3 exposure is present in the middle temperature stratum in the summer. Such a finding is probably due to changes in behaviour to avoid exposure to heat by spending more time indoors. As a result, this might lead to exposure to low levels of indoor O_3 at high temperatures and consequently the weaker relative risk for the O_3 effect.

Objective 3: Estimating changes in the acute health effects of air pollution due to changed air quality driven by climate change between the period 2065 to 2074 and the baseline 1995 to 2006

Based on the conventional method that ignores the temperature modifying effect, the largest increase in respiratory ED visits for the 2070s relative to the 2000s predicted by four GCMs was associated with O_3 exposure in the summer: approximately an additional 60 to 110 respiratory ED visits over three months. The predictions under this objective also suggest an increase, of lower magnitude compared to the estimates for O_3, in the number of respiratory ED visits due to exposure to $PM_{2.5}$ in the summer. In the winter, the predictions based on estimates derived from three of the four GCMs suggest a reduction in the number of respiratory ED visits related to exposure to $PM_{2.5}$ for the future period relative to the baseline, with a lower magnitude compared to those predicted for $PM_{2.5}$ in the summer.

An alternative method that takes account of the temperature modifying effect in estimating the impacts of climate change on air pollution-related respiratory ED visits alters the predictions made by the conventional method. In summer, the magnitude of the increased estimates of $PM_{2.5}$–respiratory ED visits in the 2070s compared to the baseline period predicted using the conventional method was greater when using the non-uniform relative risks across the temperature range. On the contrary, the excess O_3-related ED visits between the two decades predicted by the conventional method was dramatically reduced when using the alternative method. In winter, the number of $PM_{2.5}$-related respiratory ED visits in the 2070s compared to the 2000s as predicted by all the four GCMs was estimated to be increase—in a range of additional 32 to 62 respiratory ED visits over three months—when the temperature modifying effect was taken into consideration. However the results of $PM_{2.5}$-related respiratory ED visits in the winter based on the alternative method

considering the temperature modifying found in this thesis needed to be interpreted with caution due to great uncertainty in the estimates and the possibility of occurring by chance.

The extent of the difference in estimates predicted by applying the varying relative health risks across the temperature range compared to the conventional method are determined by two factors: the pattern of relative risks in the different temperature strata and the relative change in the number of days in each temperature stratum between the baseline and the future periods. A difference between the two methods for a given health outcome can be considerable if a set of relative risks applied in the alternative method is characterised by the strongest risk at extreme temperatures, either lower or upper end, with a concurrence of a considerable change in temperatures between the two periods for the temperature stratum where the strongest risk is present.

Objective 4: Assessing uncertainties of the estimations made for the health impact assessment of climate change on air pollution-related health effects

The uncertainty analysis conducted to address Objective 4 demonstrates that estimates of the health effects of air pollution are affected by non-climate related factors which potentially change in the future, as well as assumptions made in the projection methods. Among the factors and the methods examined, future population growth appears to introduce the greatest variation to the predicted changes in the future health effects of air pollution in the future period 2025 to 2034 compared to the baseline. The estimate of increased respiratory morbidity outcomes related to $PM_{2.5}$ exposure under climate change in summer and winter in combination with the growing population in the 2030s can be offset, to a large extent, by lowering air pollution emissions. The variation caused by taking into account an aging population in combination with the choice of dose-response functions is pronounced only if the health outcome predicted has a strong association with air pollution in the elderly. Assuming a 30 ppb threshold of O_3 in the summer changes the estimate of the number of respiratory morbidity outcomes with a similar magnitude of effect to the modelled future population growth in the 2030s.

9.3 Strengths and limitations of the thesis

9.3.1 Strengths

A merit of this thesis in relation to the first two objectives is the high quality of the datasets used in modelling the relationship between air pollution and acute health outcomes. Temporally, the high quality datasets were characterised by a ten-year period of time-series data which provided sufficient statistical power to detect the associations shown here. Spatially, the air pollution and weather data at fine resolution generated from the blending technique also provided a high quality exposure dataset. The blended air pollution and weather data have been shown in this thesis to be superior to measurements obtained from fixed monitors in improving the accuracy of exposures relevant to the health risks associated with air pollution.

With regard to the method applied for Objective 2, temperature cut-points that were identified to stratify temperature to detect the temperature modifying effect were not chosen arbitrarily, as has been done in past studies. Rather, the selection of temperature cut-points in this thesis involved a large number of potential candidates. In addition, the accuracy of the selected temperature cut-points was confirmed by the suitability of these temperature cut-points across multiple disease categories, health endpoints and pollutants. This can be seen from the results of sensitivity analysis conducted in Chapter 6: the temperature cut-points identified were insensitive to the changes in model specification.

In relation to Objective 3, there are three major strengths. The first strength relates to the estimations of both $PM_{2.5}$- and O_3-related health effects resulting from climate change at an urban scale. A number of studies have predicted the impacts of climate change on health burdens attributable to O_3 exposure, but only limited studies have been conducted to explore health burdens related to $PM_{2.5}$ exposure, particularly at a city level (Ebi and McGregor 2008). The second strength relates to using predictions of air pollution and temperature derived from an ensemble of four GCMs. Using an ensemble of models is widely recognised as a preferred strategy to address the uncertainty associated with climate models (Bader et al. 2008; Meehl et al. 2007). Another important strength is the use of the relative risks estimated specifically for different temperature ranges in estimating the impacts of climate change on the health effects of air pollution. This thesis demonstrates that applying the method that takes the temperature modifying effect into consideration can

potentially lead to different conclusions about the overall impact of climate change on air pollution-related health effects.

Finally, this thesis has included a careful uncertainty analysis, conducted to address Objective 4. Most studies in the past investigating the impacts of climate change on air pollution-related health effects have limited their predictions to health burdens attributable to changes in air pollution driven by only the increased GHG emissions. A number of studies have reported that uncertainties due to other factors are large and may be crucial in terms of quantifying any consequences of climate change as well as applying findings in decision making processes (Johnson and Weaver 2009). The way in which the uncertainty assessment was designed and conducted in this thesis aimed at addressing uncertainties in a systematic fashion at each stage of the prediction of the health impacts of air pollution under a changing climate.

9.3.2 Limitations

Using the blended data is not only a strength of this thesis, but can also be considered as a weakness. It was decided to use simulations of air pollution data restricted to only those days without bushfires and dust storms due to limited capacity of the modelling system employed to predict ambient air pollution concentrations associated with these events. As reported in Chapter 5, exclusion of the air pollution data on days with extreme air pollution events resulted in limited detection of a significant association between $PM_{2.5}$ exposure and mortality. In addition, producing simulations of air pollution and weather data requires high computational resources and an emissions inventory. These are not always accessible to researchers who wish to pursue epidemiological studies of air pollution.

For Objective 2, although the method used to select the temperature cut-points was robust, as discussed above, it is possible that multiple testing has resulted in some results being statistically significant by chance. The Bayesian approach recently proposed by Puza and Roberts (2013) to handle uncertainties emerging from the process of selecting the temperature cut-points may be preferable to avoid this risk.

The detection of temperature modifying effects in this thesis was limited to only respiratory morbidity outcomes in response to Objectives 3 and 4. However mortality outcomes may be of greater interest to the public and relevant authorities.

For Objective 4, due to limited computational resources in modelling future air quality under climate change, there were some uncertainties which were not considered such as those associated with different scenarios for GHG emissions and use of various climate models. Further, although the estimated changes in the air pollution-related respiratory ED visits resulting from climate change, conducted under Objective 3, compared outcomes in the 2070s with the baseline 2000s, the uncertainty analyses compared outcomes between the 2030s and the 2000s. If a similar uncertainty analysis was conducted for the 2070s, the magnitude and pattern of uncertainties associated with non-climate related factors may be different from the results presented in this thesis. In relation to the predictions made for the future health effects of $PM_{2.5}$ under this objective, another uncertainty that was not considered under this thesis is changes in PM constituents in the future. PM components in the future will be different from the present and hence the health effects associated with exposure to these PM components will also change in the future. In this thesis, this point was not addressed since the focus of the $PM_{2.5}$ and related health risks studied was on the total mass, not constituents. However this does create an additional uncertainty in the predictions.

Similar to other studies that have undertaken analysis to quantify the future impacts of climate change, it is extremely challenging to deal with future uncertainties: The further into the future that predictions are made, the greater the level of uncertainty that exists. Furthermore, these uncertainties when considered together may exhibit a cumulative effect, or Gosling et al. (2009) referred to it as a "cascading effect" in their review paper on a rise in temperature and potential impact on mortality. As reported in this thesis, addressing potential future uncertainties to make the quantified risk estimates more transparent and informative is generally a trade-off to the time and financial resources exploited. Therefore only a limited number of uncertainties can be included and considered.

9.4 Applications and policy implications of thesis findings

9.4.1 Application of thesis findings

One explicit application of the findings from this thesis is that the temperature stratum-specific relative risks estimated in Chapter 6 were able to be used to refine the estimates of the impacts of climate change on air pollution-related health in Chapter 8. In the context of climate change, mobilisation of the knowledge and research outcomes from past studies in the area of the temperature modifying effect on the relationships between air pollution and

health is still limited. This thesis has highlighted that, in addition to using the conventional method relying on an invariable risk across different temperatures to predict future changes in the health effects of air pollution, considering the temperature modifying effect would be of particular importance in a warmer world. Application of the variable relative risks could lead to better accuracy of estimates of the future health risks of air pollution on hot days. Such improved estimates will be more informative for planning and decision making to avoid the extra health burdens caused by simultaneous exposure to heat and air pollution.

9.4.2 Policy implications

Implications of the thesis findings in this section are discussed in relation to two facets: the temperature modifying effect explored in response to Objective 2 and the estimated air pollution-related health effects due to climate change obtained from addressing Objective 3 and Objective 4.

9.4.2.1 Temperature modifying effect

Despite limited scientific evidence to confirm the temperature modifying effect, a course of appropriate actions should be prepared for handling potential exacerbations of ill health due to exposure to air pollution under extreme temperature episodes. From a perspective of protecting health from exposure to extreme heat, in some cities particularly those that have experienced heatwave events such as Melbourne, preparedness including heat health alert systems are already place (Victorian Department of Health 2012). The preparedness and plans to prevent the adverse health impacts from exposure to heat are operated under the auspices of health agencies. From a perspective of minimising harm in populations from air pollution exposure, alert and early warning systems when air pollution concentrations go beyond the safety level are generally implemented under the responsibility of environmental agencies. Clearly these two systems have been run to cope with these two environmental factors in separate domains.

To respond to the possibility of an increased health risk associated with air pollution on hot days, an integrated system might be needed in the future with coordination from both government agencies. Given that some strategies to cope with these two environmental hazards and populations at risk are in common, such a new integrated system might bring mutual benefits for the protection of health from exposure to air pollution and heat. One

example is that thresholds of air pollution concentrations currently in place may have to be lowered on hot days.

9.4.2.2 Estimated air pollution-related health impacts of climate change

The estimated changes in air pollution-related health effects due to climate change as presented in this thesis could be incorporated into cost-benefit analysis. The estimated health impacts, after being translated into monetary value, can provide evidence to guide actions and measures outside and inside public health sectors. For example Knowlton et al. (2008) reported that the results of estimating changes in heat and O_3-related mortality under a changing climate in New York City were requested by the city council for making decisions on laws to regulate GHG emissions.

The results of the uncertainty assessment can be used to guide the management of the health risks of air pollution under climate change. Consideration can be given to reducing air pollution emissions, avoiding exposure to air pollution and targeting populations at risk.

Firstly, there are some actions that can be taken locally to deal with the potential of increased air pollution concentrations induced by climate change and health consequences including reduction of air pollution emissions. This thesis reports that the changing climate could have potential impacts on future air quality in the PPR, to a large extent in the 2070s and to a lesser extent in the 2030s. The changes in air quality are likely to increase $PM_{2.5}$ and O_3-related health effects particularly in the summer. Of course, such an additional health effect associated with $PM_{2.5}$ and O_3 exposure estimated for the future in this thesis due to climate change can be avoided through actions to cut GHG emissions. Climate change is a global issue which requires cumulative actions made by the nations around the world to curb the rising GHGs emissions. An assessment of progress to date in limiting the GHG levels has indicated that it is still feasible to achieve the below 2°C target set by the international community only if immediate and strong actions are taken internationally particularly by industrialised countries (UNEP 2012).

Local reduction in air pollution emissions is another key measure to manage the undesirable impacts of climate change on air quality. As demonstrated in the estimates made under Groups C1 and C2 of Chapter 8, the impact of climate change-induced elevations of air pollution concentrations (and consequent health burdens) to the 2030s could be largely offset by reduction of air pollution emissions. In addition, the reduction of

air pollution locally provides an ancillary-benefit to the reduction of GHGs globally. Several air pollutants for example black carbon, O_3 and VOCs are GHGs. Thus the reduction of these air pollutants indirectly results in slowing down climate change impacts. Furthermore, since most air pollutants arise from many of the same sources of GHGs, targeting mitigation of air pollution at a local level, can, to a large extent, ameliorate increase in GHGs and hence limit the associated health risks. A number of studies have proposed policies that can both reduce air pollution and provide ancillary benefits to climate-change mitigation as a promising way forward (Cifuentes et al. 2001; Nemet et al. 2010; Haines et al. 2009; Aunan et al. 2006).

Exposure to air pollution can be achieved through behavioural changes and adaptation. As demonstrated in Section 8.4, although the O_3 concentrations in the 2070s were predicted to increase considerably due to the increased temperature, the estimated changes in O_3-related respiratory ED visits in the summer, taking into account the temperature modifying effect, were predicted to be minimal. This was due to the weak relative risk associated with exposure to O_3 in the summer for this respiratory outcome estimated on hot days. Such a weak relative risk may be related to behavioural change to avoid heat exposure. Therefore public health interventions that can alter behaviour of people to avoid exposure to heat, for example using air conditioners and staying indoors, might have a co-benefit of reducing health burdens related to exposure to air pollution. It might be argued that spending more time indoors may lead to other potential health costs such as cardiovascular diseases induced by physical inactivity. Therefore such an intervention would have to be implemented with a great care to prevent additional side effects on health. For example a recommendation to stay indoors on hot days in summer could be more specific in that individuals may choose to exercise indoors rather than having outdoor activities, to avoid heat and air pollution exposure.

Lastly, sensitive populations can be targeted through managing factors which can shift dose-response functions to a lower level. In the health impact function (Equation 1.1 introduced in Chapter 1) used in estimating the impacts of climate change on the health effects of air pollution in this thesis, the dose-response function is a key variable in determining the magnitude of changes in the effects. Evidence derived from past studies suggests that the dose-response function can be modified by a number of factors including pre-existing health conditions, the health endpoint under consideration, socioeconomic status, age, season, and temperature (Medina-Ramon and Schwartz 2008; Park et al. 2011;

Chen et al. 2007a; Charafeddine and Boden 2008). To alleviate the air pollution-related health impacts under climate change, public health interventions such as preventing obesity in the population (Chen et al. 2007a) which have potential to lower health risks associated with exposure to air pollution can also help reduce the health burdens resulting from climate change in the future. As demonstrated in the estimates made under Group E, the variation introduced by the choice of dose-response functions was larger than that associated with the impact of climate change.

9.5 Potential for future research

In relation to Objective 2, the results reported here and from other epidemiological studies have provided evidence to support the variation in risk estimates associated with air pollution across the temperature range. Greater health risks due to air pollution exposure have largely been evident at both ends of the temperature range. Despite a growing body of evidence from epidemiological studies, more evidence from human and animal toxicological studies is needed to confirm plausibility as well as to explore mechanisms. Bringing together evidence from epidemiological studies and studies of animal and human toxicology will be an important step further to answer whether or not concurrent exposure to air pollution and extreme temperatures can bring an additional threat to population health, in particular under a warmer world.

As speculated in Section 6.4.3, the greater risk estimate for respiratory ED visits associated with O_3 in summer in the middle temperature stratum compared to the other temperature strata may be due to how people respond to meteorological conditions. To confirm such speculation, it would be useful to have an Australian study about behaviours in hot weather and/or during high levels of pollution. This kind of study could be useful to help formulate an intervention to prevent health impacts from simultaneous and/or separate exposure to heat and air pollution on hot days and on days with air pollution episodes.

This thesis has left the issue of possible non-linear relationships between exposure to $PM_{2.5}$ total mass and mortality unanswered. Research on this issue is currently active and more information gained from the growing body of research, particularly from North America, may provide more insight into the relationship between the chemical components of $PM_{2.5}$ and health effects (Bell 2012).

Recommendations for future research in the area of estimating air pollution-related health and climate change are discussed as follows.

- Predicting $PM_{2.5}$ in response to climate change and health consequences

As can be seen from the results in Chapter 8, the health impacts of $PM_{2.5}$ predicted to be altered under a changing climate could be as large as those related to O_3. However there have been fewer studies attempting to predict the impact of climate change on $PM_{2.5}$ compared to O_3. Future research that investigates changes in $PM_{2.5}$ undertaken at the regional and local levels would be particularly valuable as responses of $PM_{2.5}$ to global meteorological conditions are highly variable in different locations. Using air quality modelling, it should be possible to track the individual contributions of sea salt, dust, vehicle exhaust etc to total $PM_{2.5}$ concentrations and exposure, which may assist in determining associations with health end-points.

- Taking into account the temperature modifying effect

Evidence based on a considerable number of epidemiological studies has suggested that the health effects of air pollution can be modified by temperature. Nonetheless integrating the knowledge in this area in the context of health impacts of climate change is lacking. Therefore future research should consider the modification effect of temperature to estimate the health impacts of air pollution driven by climate change.

- Uncertainty analysis

The results of this thesis suggest that estimates of changes in the health impacts of air pollution under a changing climate are subject to large variation. Therefore uncertainty analysis should be a standard approach for future studies estimating the impacts of climate change on any health risks. Undoubtedly the uncertainties regarding climate modelling systems and scenarios of GHG emissions should be assessed in any fields that climate change might have impacts on. Specific to estimations of the health impacts related to air pollution, the analyses here lead to a recommendation that four factors at least should be included in the uncertainty analysis: population growth, changes in age structure, changes in air pollution emissions and the choice of dose-response functions. For estimating changes in the O_3 effect resulting from climate change, as long as the knowledge in the

area of O_3 thresholds is inconclusive, analysis of this factor should also be considered as part of the evaluation of uncertainties.

Since the uncertainty assessment conducted in this thesis involved a modest number of factors, a simple quantitative method for comparing changes in health impacts between the factors was appropriate. However a better quantitative method, for example ANOVA, would be desirable when a large number of factors in the uncertainty assessment need to be addressed, as was done in two recent studies (Post et al. 2012; Wu et al. 2013).

- Assess the health impacts of extreme air pollution episodes under climate change

Assessing the impact of climate change on forest fires will be increasingly important particularly for a country like Australia where forest fires are common. Evidence drawn from past studies indicates that the smoke from forest fires is harmful to health (Mott et al. 2005; Analitis et al. 2011). There is also a possibility that climate change may cause increases in the occurrence of extreme air pollution episodes including forest fires (Flannigan et al. 2009; Aldersley et al. 2011; Hughes and Steffen 2013). Current research into projecting future forest fire activity resulting from climate change could be used downstream to benefit research in public health.

Although climate change is likely to alter the health risks of air pollution, there is an array of other health problems including temperature-related health effects, disaster-related health losses, vector-borne diseases, water-borne diseases and mental illnesses that may also be potentially affected (McMichael 2013) . Thus, future analyses to quantify health risks from climate change could rank air pollution issues against other potential problems. Doing so might help decision makers decide what issues need most investment now.

9.6 Concluding remarks

In conclusion, this thesis demonstrates the potential in advancing the whole stream of research on the impacts of climate change on air pollution-related health—a research area that is still at an early stage of development.

At the upstream of this research area, this thesis begins with exploring another function and additional benefit of the use of simulated air pollution and weather data, which are

generated from the modelling system predicting future air pollution concentrations and weather, in the epidemiological element of this thesis. Such functions of air quality modelling enable this thesis to obtain more accurate dose-response functions which are subsequently used in the health impact models for predicting future health risks associated with air pollution in the context of climate change.

At the middle stream, this thesis adds evidence of the existence of temperature modifying effects on the association of air pollution with health outcomes in the Melbourne Region. The evidence of the temperature modifying effect acts as a spring board for this thesis to incorporate this important aspect for the purpose of quantifying the future health impacts of air pollution induced by climate change.

At the downstream, estimations of changes in air pollution-related respiratory ED visits made in this thesis for the medium-term future in the Melbourne Region suggest that the pollutant of most concern is O_3, and the levels are likely to increase and cause additional health burdens in the summer. By applying different relative risks across the temperature range, findings from this thesis suggest that estimations of changes in the health risks obtained might be considerably different from those relying on the conventional method. This emphasises that this issue should be addressed in future studies investigating the health effects of air pollution under a changing climate. Finally, this thesis highlights the importance of performing uncertainty assessment as the findings from the analyses in this thesis reveal that variation due to changes in some factors in the future and projection methods might be larger than that of the impact of climate change itself.

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Appendices A–Appendices in Chapter 3

Appendix A.1 Twenty two ambient air monitoring stations in Victoria.

Note: Red dots represent 14 stations located inside SD 205 and black dots are 8 remote stations located outside SD 205

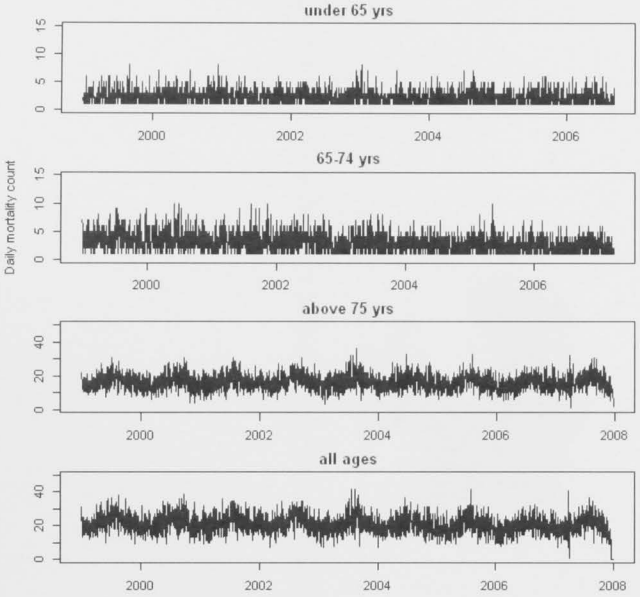
Appendix A.2 Changes to SLA codes during 1999 to 2008

SLA in 1999 to 2004	Change to SLAs in 2005 to 2008
7262 *	7267
7265*	7261
7268*	7264
7074	7075 and 7076
3671	3672 and 3673
7455	7452, 7453 and 7456

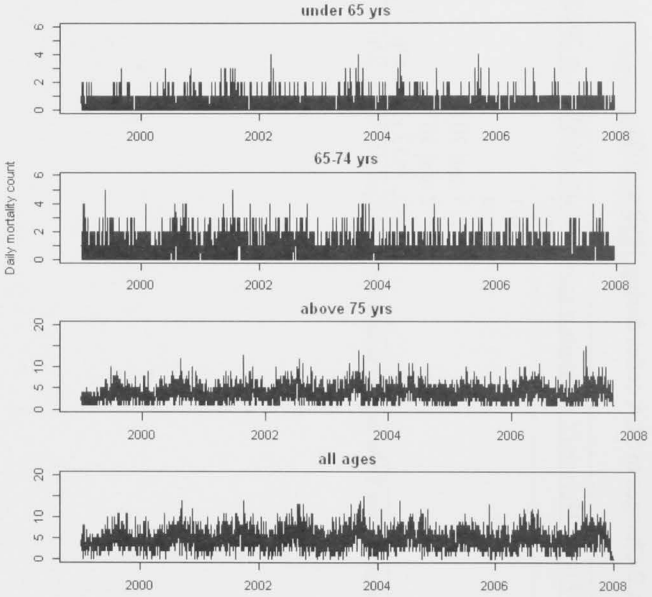
Note * the change in these SLAs were applicable only for the period 1999 to 2000

Appendix A.3 Time-series plots of cardiovascular and respiratory mortality by age, January 1999 to December 2007

a) Cardiovascular mortality



b) Respiratory mortality



Appendix A.4 Exceedences for O_3, PM_{10} and $PM_{2.5}$ due to (a) bushfires and (b) windblown, 1999 to 2008

a) Bushfire

Year	Pollutant	Date
1999	PM_{10}	Jul: 15
2001	PM_{10}	Jan: 12
2003	O_3	Jan : 17, 24, 25
		Feb: 3
	PM_{10}	Jan: 19, 20, 24, 25, 29
		Feb: 2, 3, 4, 5, 20
	$PM_{2.5}$	Jan: 25
		Feb: 3, 6
2006	O_3	Jan: 25
		Dec: 9, 20, 30
	PM_{10}	Jan: 25, 26, 27
		Nov: 30
		Dec: 5, 8, 9, 10, 11, 13, 14, 20, 21, 22, 30, 31
	$PM_{2.5}$	Jan: 27
		Dec: 14, 20
2007	O_3	Jan: 9
	PM_{10}	Jan: 9, 10, 16, 17
		Apr: 10, 13, 14, 19
	$PM_{2.5}$	Jan: 1, 10
2008	PM_{10}	Apr: 19, 20, 23, 24, 25
	$PM_{2.5}$	Apr: 19, 25
		May: 13

b) Windblown dust

Year	Pollutant	Date
2002	PM_{10}	Oct: 18 (d)
		Nov: 8 (d),12 (d), 13 (d), 14 (d)
		Dec: 29 (ds)
2003	PM_{10}	Jan: 13 (d)
		Mar: 18 (d), 19 (ds)
		Jun: 5 (d), 6 (ds)
		Nov: 15 (d)
2004	PM_{10}	Jan: 20 (d), 28 (d)
		Feb: 20 (d)
		Mar: 4 (d), 25 (d), 28 (d)
		Apr: 14 (d), 22(d)
		May: 15 (d)
		Dec: 6(d), 24(d)
2005	PM_{10}	Apr: 1, 3, 28
2006	PM_{10}	Jan: 20 (d)
		Feb: 8 (d)
		Apr: 11 (d)
		Sep: 1 (d)
		Oct: 12 (d), 19 (d), 21 (d)
		Nov: 11 (d), 21 (d)
2007	PM_{10}	Jan: 31(d)
		Feb: 2 (d), 5 (d)
		Mar: 7 (d), 23 (d)
		Aug: 30 (d), 31 (d)
		Oct: 3 (d), 19 (d), 20 (d), 21 (d)
2008	PM_{10}	Jan: 11 (d)
		Mar: 14 (d),15 (d)
		Apr: 2 (d)
		Oct: 18 (d), 29 (d), 30 (d)
		Nov: 13 (d)

Note: d=dust, ds=dust storm

Source: EPA Victoria (1999-2008)

Appendix A.5 Pairwise correlation of air quality and weather variables for All-seasons and by season

a) All-seasons

	Bsp	Partisol $PM_{2.5}$	Combined $PM_{2.5}$	O_3 max 1-h	O_3 max 4-h	O_3 max 8-h	O_3 24-h	Temp max	Temp min	Temp average	Dew point
Bsp	1.00										
Partisol $PM_{2.5}$	0.82	1.00									
Combined $PM_{2.5}$	1.00	0.85	1.00								
O_3 max 1-h	0.28	0.08	0.27	1.00							
O_3 max 4-h	0.24	0.03	0.24	0.99	1.00						
O_3 max 8-h	0.19	-0.04	0.18	0.96	0.98	1.00					
O_3 24-h	0.01	-0.20	0.01	0.75	0.80	0.86	1.00				
Temp max	0.15	0.08	0.14	0.71	0.70	0.69	0.48	1.00			
Temp min	-0.03	-0.13	-0.03	0.39	0.39	0.42	0.43	0.72	1.00		
Temp average	0.07	-0.03	0.07	0.63	0.63	0.64	0.51	0.95	0.88	1.00	
Dew point	0.07	0.00	0.07	0.29	0.27	0.25	0.12	0.59	0.79	0.70	1.00

b) Summer

	Bsp	Partisol $PM_{2.5}$	Combined $PM_{2.5}$	O_3 max 1-h	O_3 max 4-h	O_3 max 8-h	O_3 24-h	Temp max	Temp min	Temp average	Dew point
Bsp	1.00										
Partisol $PM_{2.5}$	0.91	1.00									
Combined $PM_{2.5}$	1.00	0.92	1.00								
O_3 max 1-h	0.47	0.48	0.47	1.00							
O_3 max 4-h	0.47	0.48	0.46	0.99	1.00						
O_3 max 8-h	0.46	0.48	0.46	0.97	0.98	1.00					
O_3 24-h	0.41	0.46	0.40	0.84	0.87	0.91	1.00				
Temp max	0.25	0.33	0.25	0.81	0.81	0.81	0.71	1.00			
Temp min	0.11	0.28	0.11	0.34	0.35	0.38	0.43	0.54	1.00		
Temp average	0.22	0.34	0.22	0.73	0.74	0.76	0.70	0.94	0.75	1.00	
Dew point	0.06	0.10	0.06	0.27	0.25	0.24	0.14	0.33	0.67	0.46	1.00

c) Autumn

	Bsp	Partisol $PM_{2.5}$	Combined $PM_{2.5}$	O_3 max 1-h	O_3 max 4-h	O_3 max 8-h	O_3 24-h	Temp max	Temp min	Temp average	Dew point
Bsp	1.00										
Partisol $PM_{2.5}$	0.83	1.00									
Combined $PM_{2.5}$	1.00	0.85	1.00								
O_3 max 1-h	0.22	0.15	0.22	1.00							
O_3 max 4-h	0.17	0.09	0.17	0.99	1.00						
O_3 max 8-h	0.07	-0.03	0.07	0.95	0.98	1.00					
O_3 24-h	-0.15	-0.26	-0.15	0.71	0.76	0.84	1.00				
Temp max	0.11	0.07	0.11	0.76	0.76	0.73	0.50	1.00			
Temp min	-0.21	-0.34	-0.21	0.37	0.39	0.46	0.54	0.60	1.00		
Temp average	-0.05	-0.15	-0.05	0.66	0.67	0.70	0.59	0.92	0.84	1.00	
Dew point	-0.01	-0.11	-0.02	0.26	0.24	0.25	0.15	0.45	0.70	0.60	1.00

d) Winter

	Bsp	Partisol $PM_{2.5}$	Combined $PM_{2.5}$	O_3 max 1-h	O_3 max 4-h	O_3 max 8-h	O_3 24-h	Temp max	Temp min	Temp average	Dew point
Bsp	1.00										
Partisol $PM_{2.5}$	0.81	1.00									
Combined $PM_{2.5}$	1.00	0.85	1.00								
O_3 max 1-h	-0.53	-0.52	-0.53	1.00							
O_3 max 4-h	-0.56	-0.54	-0.56	0.99	1.00						
O_3 max 8-h	-0.58	-0.56	-0.58	0.95	0.98	1.00					
O_3 24-h	-0.61	-0.58	-0.62	0.81	0.84	0.90	1.00				
Temp max	-0.01	-0.01	-0.01	0.06	0.06	0.02	-0.05	1.00			
Temp min	-0.45	-0.48	-0.45	0.11	0.14	0.21	0.37	0.34	1.00		
Temp average	-0.35	-0.36	-0.35	0.14	0.16	0.19	0.26	0.76	0.84	1.00	
Dew point	0.02	-0.11	0.02	-0.22	-0.23	-0.20	-0.12	0.23	0.60	0.51	1.00

e) Spring

	Bsp	Partisol $PM_{2.5}$	Combined $PM_{2.5}$	O_3 max 1-h	O_3 max 4-h	O_3 max 8-h	O_3 24-h	Temp max	Temp min	Temp average	Dew point
Bsp	1.00										
Partisol $PM_{2.5}$	0.58	1.00									
Combined $PM_{2.5}$	0.99	0.66	1.00								
O_3 max 1-h	0.43	0.09	0.42	1.00							
O_3 max 4-h	0.39	0.07	0.39	0.99	1.00						
O_3 max 8-h	0.32	0.03	0.32	0.95	0.98	1.00					
O_3 24-h	0.02	-0.13	0.01	0.62	0.67	0.76	1.00				
Temp max	0.35	0.10	0.34	0.71	0.69	0.66	0.38	1.00			
Temp min	0.07	0.00	0.07	0.22	0.21	0.21	0.36	0.52	1.00		
Temp average	0.25	0.06	0.25	0.61	0.59	0.58	0.43	0.93	0.76	1.00	
Dew point	0.27	0.02	0.26	0.12	0.07	0.00	-0.06	0.33	0.68	0.49	1.00

Appendices B–Appendices in Chapter 4

Appendix B.1 Optimal length scales with the unit in km (in x- and y- directions) of each variable separated by season

Variable	All-seasons	Autumn	Spring	Summer	Winter
O_3 24-h	30,30	30,30	100,50	30,30	40,30
O_3 max 1-h	30,30	30,30	60,50	30,30	40,30
O_3 max 4-h	30,30	30,30	100,50	30,30	40,30
O_3 max 8-h	30,30	30,30	90,50	30,30	40,30
$PM_{2.5}$ 24-h	20,40	20,40	20,50	20,40	30,30
Temp 24-h	30,90	30,90	30,80	30,70	40,70
Temp max 1-h	30,70	30,50	30,40	30,70	30,50
Temp min 1-h	40,80	30,70	30,70	30,40	30,50
Dew 24-h	50,50	40,50	50,40	40,50	50,40
Dew max1-h	40,80	40,40	40,70	40,90	50,80
Dew min 1-h	30,60	40,50	40,50	30,50	50,50

Appendix B.2 Definitions and interpretations of model performance indicators

Where M and O are modelled and observed concentrations for each pair i respectively, $\bar{M}$ and $\bar{O}$ are the mean modelled and observed concentrations respectively, and N is the total number of modelled/observed pairs, each indicator is defined as below (USEPA 2005; Thunis et al. 2011);

1. Mean Bias (MB) :

$$MB = \frac{1}{N}\sum_{i=1}^{N}(M_i - O_i)$$

An ideal mean bias of zero indicates that the model over-predictions and model under-predictions exactly cancel each other out.

2. Gross Error (GE):

$$GE = \frac{1}{N}\sum_{i=1}^{N}|M_i - O_i|$$

It is similar to mean bias except that the absolute value of the difference is used so that the error is always positive.

3. Fractional Bias (FB)

$$FB = \left(\frac{1}{N}\sum_{i=1}^{N}\frac{M_i - O_i}{(M_i + O_i)/2}\right) * 100\%$$

Given that M and O are always positive, Fractional Bias ranges from -200% to 200%. This indicator normalises the bias for each modelled-observed pair by the average of the model and observation. FB has the advantage of bounding the maximum bias and error and does not allow a few data points to dominate the matrix. This indicator can be especially useful in assessing performance of PM modelling where species might exhibit values close to zero.

4. Fractional Error (FE)

$$FE = \left(\frac{1}{N} \sum_{i=1}^{N} \frac{|M_i - O_i|}{(M_i + O_i)/2} \right) * 100\%$$

Fractional Error ranges from 0% to 200%. This indicator is similar to FB in that it gives equal weight to under- and over-prediction. Therefore it is not sensitive to a threshold in measured values and does not assume that observations are the truth.

5. Correlation (R):

$$R = \frac{\sum_{i=1}^{N}(M_i - \bar{M})(O_i - \bar{O})}{\sqrt{\sum_{i=1}^{N}(M_i - \bar{M})^2} \sqrt{\sum_{i=1}^{N}(O_i - \bar{O})^2}}$$

R ranges from -1 to +1 and indicates the strength of a linear relationship between the two datasets. A value of +1 corresponds to all the pairs lying on a straight line with the positive slope in the scatter diagram. A value of *R* near zero indicates the absence of linear correlation between the variables.

6. Root Mean Square Error (RMSE):

$$RMSE = \sqrt{\frac{1}{N} \sum_{i=1}^{N} (M_i - O_i)^2}$$

The ideal value is zero. The RMSE is a good overall measure of model performance. However, since large errors are weighed heavily (due to squaring), few large errors may produce a large RMSE even though the errors may be small and quite acceptable elsewhere.

7. Index of Agreement (IOA):

$$IOA = 1 - \frac{\sum_{i=1}^{N}(M_i - O_i)^2}{\sum_{i=1}^{N}(|M_i - \bar{O}| + |O_i - \bar{O}|)^2}$$

The perfect value of IOA is 1. IOA determines the extent to which magnitudes of observed mean ($\bar{O}$) are related to the predicted deviations about $\bar{O}$, and allows for sensitivity toward differences in O and M.

Appendix B.3 Performance indicators between observed and fully blended air pollution data over the period 1999 to 2008 in the Melbourne Region

Air pollution variable	No. of days	Observed mean	Blended mean	MB	GE	FB (%)	FE (%)	R	RMSE	IOA
O_3 24-h (ppb)										
All-seasons	3571	14.6	14.9	0.3	0.6	3.8	5.5	0.993	0.8	0.995
Autumn	908	12.5	12.8	0.4	0.6	4.6	6.6	0.989	0.8	0.993
Spring	905	17.5	17.7	0.2	0.6	1.7	3.7	0.990	0.8	0.994
Summer	840	16.4	16.7	0.3	0.5	1.9	3.7	0.994	0.8	0.996
Winter	918	12.2	12.6	0.4	0.6	6.8	8.0	0.994	0.8	0.995
O_3 max 1-h (ppb)										
All-seasons	3571	27.0	27.1	0.1	0.5	0.4	1.9	0.998	0.7	0.999
Autumn	908	25.4	25.4	0.0	0.5	0.1	2.1	0.997	0.7	0.999
Spring	905	28.8	28.9	0.1	0.4	0.4	1.6	0.997	0.6	0.998
Summer	840	31.6	31.6	0.0	0.5	0.2	1.8	0.998	0.8	0.999
Winter	918	22.7	22.9	0.2	0.4	1.0	2.2	0.994	0.6	0.997
O_3 max 4-h (ppb)										
All-seasons	3573	25.2	25.3	0.1	0.5	0.6	2.1	0.998	0.7	0.999
Autumn	908	23.6	23.7	0.0	0.5	0.2	2.4	0.997	0.7	0.998
Spring	907	27.3	27.4	0.1	0.4	0.4	1.6	0.997	0.5	0.998
Summer	840	29.3	29.3	0.0	0.5	0.3	1.9	0.998	0.7	0.999
Winter	918	21.2	21.4	0.2	0.4	1.4	2.6	0.995	0.6	0.997
O_3 max 8-h (ppb)										
All-seasons	3573	23.0	23.1	0.2	0.5	1.1	2.6	0.997	0.7	0.998
Autumn	908	20.8	20.9	0.2	0.5	1.1	3.0	0.996	0.8	0.998
Spring	907	26.0	26.0	0.1	0.4	0.4	1.8	0.996	0.6	0.998
Summer	840	26.9	26.9	0.1	0.5	0.3	2.0	0.998	0.7	0.999
Winter	918	18.5	18.8	0.3	0.5	2.5	3.6	0.995	0.7	0.997
$PM_{2.5}$ 24-h ($\mu g/m^3$)										
All-seasons	3545	7.0	6.5	-0.5	0.6	-7.9	8.1	0.993	0.8	0.990
Autumn	897	8.1	7.3	-0.7	0.8	-9.6	10.0	0.993	1.0	0.987
Spring	897	5.9	5.5	-0.3	0.3	-5.9	6.1	0.991	0.4	0.989
Summer	836	6.5	6.0	-0.5	0.5	-7.3	7.4	0.997	0.6	0.991
Winter	915	7.7	7.0	-0.6	0.7	-8.6	8.9	0.993	0.9	0.990

Appendix B.4 Performance indicators between observed and fully blended weather data over the period 1999 to 2008 in the Melbourne Region

Weather variable (°C)	No. of days	Observed mean	Blended mean	MB	GE	FB (%)	FE (%)	R	RMSE	IOA
Temp 24-h										
All-seasons	3619	14.7	14.8	0.1	0.1	0.4	0.5	1.000	0.1	1.000
Autumn	918	15.2	15.3	0.0	0.1	0.3	0.4	1.000	0.1	1.000
Spring	910	14.4	14.4	0.1	0.1	0.5	0.6	1.000	0.1	1.000
Summer	872	19.3	19.4	0.1	0.1	0.6	0.6	1.000	0.1	1.000
Winter	919	10.3	10.3	0.0	0.1	0.2	0.5	1.000	0.1	1.000
Temp max 1-h										
All-seasons	3619	19.8	19.9	0.1	0.1	0.6	0.7	1.000	0.2	1.000
Autumn	918	20.3	20.4	0.1	0.1	0.6	0.7	1.000	0.2	1.000
Spring	910	19.5	19.7	0.2	0.2	0.8	0.8	1.000	0.2	1.000
Summer	872	25.4	25.5	0.1	0.2	0.5	0.6	1.000	0.2	1.000
Winter	919	14.1	14.1	0.0	0.1	0.4	0.6	0.999	0.1	1.000
Temp min 1-h										
All-seasons	3619	10.1	10.1	0.0	0.1	-0.2	1.1	1.000	0.1	1.000
Autumn	918	10.6	10.6	0.0	0.1	0.0	0.9	0.999	0.1	1.000
Spring	910	9.3	9.3	0.0	0.1	0.3	1.0	0.999	0.1	1.000
Summer	872	13.9	13.9	0.0	0.1	-0.2	0.6	0.999	0.1	1.000
Winter	919	6.8	6.7	0.0	0.1	-0.9	1.7	0.999	0.1	1.000
Dew 24-h										
All-seasons	3611	8.1	8.1	0.0	0.0	-0.1	0.7	1.000	0.1	1.000
Autumn	918	8.9	8.9	0.0	0.0	0.0	0.6	1.000	0.1	1.000
Spring	910	7.2	7.2	0.0	0.0	-0.1	1.1	1.000	0.1	1.000
Summer	865	11.0	11.0	0.0	0.0	-0.1	0.4	1.000	0.1	1.000
Winter	918	5.7	5.6	0.0	0.0	-0.2	0.8	1.000	0.1	1.000
Dew max 1-h										
All-seasons	3611	10.9	10.9	0.0	0.1	0.0	0.7	1.000	0.1	1.000
Autumn	918	11.7	11.7	0.0	0.1	-0.1	0.5	1.000	0.1	1.000
Spring	910	10.2	10.2	0.0	0.1	0.0	0.8	0.999	0.1	1.000
Summer	865	14.1	14.1	0.0	0.1	0.0	0.5	0.999	0.1	1.000
Winter	918	7.9	7.9	0.0	0.1	0.1	0.9	0.999	0.1	0.999
Dew min 1-h										
All-seasons	3611	5.6	5.6	0.0	0.1	-0.5	4.6	0.999	0.1	1.000
Autumn	918	6.4	6.4	0.0	0.1	-0.6	1.8	0.999	0.1	1.000
Spring	910	4.5	4.5	-0.1	0.1	-2.1	7.5	0.999	0.1	1.000
Summer	865	8.1	8.0	0.0	0.1	-0.9	2.6	0.999	0.1	1.000
Winter	918	3.6	3.6	0.0	0.1	1.6	6.5	0.999	0.1	1.000

Appendix C.1 Estimates of percentage changes (95% CI) in daily mortality derived from the standard approach, presented by disease categories, age groups, the single- or two-pollutant models, and seasons per 10 $\mu g/m^3$ increase for $PM_{2.5}$

	Effect of $PM_{2.5}$ on mortality					
	All-seasons		Summer		Winter	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
All-cause						
All-ages	-0.082	-0.121	-0.175	1.809	-1.318	1.302
	(-1.810, 1.649)	(-1.887, 1.648)	(-6.604, 6.296)	(-5.279, 8.948)	(-4.360, 1.734)	(-3.341, 5.966)
<65 yrs	-2.743	-3.231	-4.887	0.093	-4.333	8.218
	(-6.990, 1.523)	(-7.566, 1.124)	(-19.876, 10.330)	(-16.465, 16.931)	(-11.840, 3.230)	(-3.385, 19.956)
65–74 yrs	1.583	1.126	-0.631	7.665	2.913	6.336
	(-2.600, 5.783)	(-3.161, 5.431)	(-16.513, 15.508)	(-9.801, 25.438)	(-4.512, 10.393)	(-5.085, 17.888)
≥75 yrs	0.149	0.339	1.352	0.743	-1.658	-1.605
	(-1.970, 2.273)	(-1.825, 2.508)	(-6.592, 9.360)	(-8.013, 9.575)	(-5.377, 2.075)	(-7.246, 4.069)
Cardiovascular						
All-ages	0.001	0.038	1.317	0.065	-3.150	0.722
	(-2.823, 2.833)	(-2.856, 2.940)	(-9.579, 12.334)	(-11.902, 12.177)	(-8.033, 1.757)	(-6.758, 8.258)
<65 yrs	-5.285	-3.620	-0.233	-1.286	-14.568	-3.172
	(-14.632, 4.151)	(-13.127, 5.979)	(-32.854, 33.488)	(-36.910, 35.656)	(-30.966, 2.107)	(-27.989, 22.279)
65–74 yrs	3.623	1.672	18.442	28.966	6.345	2.702
	(-4.133, 11.438)	(-6.327, 9.736)	(-12.223, 50.059)	(-4.769, 63.845)	(-7.368, 20.248)	(-18.309, 24.163)
≥75 yrs	-0.006	0.169	-1.034	-4.406	-3.412	0.754
	(-3.208, 3.206)	(-3.112, 3.460)	(-13.518, 11.609)	(-18.123, 9.503)	(-8.923, 2.131)	(-7.688, 9.269)
Respiratory						
All-ages	-1.441	-0.766	-5.567	-8.784	-0.041	-3.938
	(-7.332, 4.486)	(-6.805, 5.308)	(-28.682, 18.098)	(-34.228, 17.329)	(-10.088, 10.107)	(-19.071, 11.428)
<75 yrs	6.060	6.529	-6.949	2.426	0.970	22.019
	(-6.123, 18.393)	(-6.031, 19.247)	(-57.875, 46.729)	(-54.044, 62.266)	(-19.721, 22.097)	(-10.209, 55.298)
≥75 yrs	-3.742	-2.977	-4.334	-11.286	-0.372	-11.990
	(-10.466, 3.027)	(-9.856, 3.950)	(-30.286, 22.312)	(-39.796, 18.071)	(-11.862, 11.252)	(-29.112, 5.435)

Appendix C.2 Estimates of percentage changes (95% CI) in daily mortality derived from the standard approach, presented by disease categories, age groups, the single- or two-pollutant models, and seasons per 10 ppb increase for O_3

	Effect of O_3 on mortality			
	All-seasons		Summer	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
All-cause				
All-ages	-0.595	-0.760	-1.371	-1.811
	(-1.942 ,0.755)	(-2.136, 0.619)	(-3.817, 1.080)	(-4.530, 0.915)
<65 yrs	-0.849	-1.076	-4.578	-4.618
	(-4.116, 2.429)	(-4.412, 2.272)	(-10.415, 1.293)	(-11.108, 1.915)
65–74 yrs	-3.091	-2.850	-5.686	-7.378
	(-6.314, 0.144)	(-6.141, 0.451)	(-11.445, 0.107)	(-13.734, -0.981)
≥75 yrs	0.153	-0.090	0.749	0.558
	(-1.511, 1.820)	(-1.791, 1.613)	(-2.297, 3.805)	(-2.834, 3.962)
Cardiovascular				
All-ages	1.483	1.421	1.025	1.137
	(-0.745, 3.716)	(-0.858, 3.704)	(-3.064, 5.131)	(-3.402, 5.697)
<65 yrs	6.615	7.017	-0.011	1.031
	(-0.621, 13.903)	(-0.387, 14.476)	(-13.000, 13.149)	(-13.321, 15.591)
65–74 yrs	-2.106	-2.180	-4.615	-9.306
	(-8.182, 4.007)	(-8.384, 4.063)	(-15.553, 6.445)	(-21.312, 2.848)
≥75 yrs	1.480	1.369	2.181	3.044
	(-1.057, 4.022)	(-1.226, 3.970)	(-2.506, 6.890)	(-2.171, 8.286)
Respiratory				
All-ages	1.958	0.610	1.303	2.897
	(-2.713, 6.650)	(-4.176, 5.420)	(-7.483, 10.167)	(-6.880, 12.770)
<75 yrs	-2.138	-2.067	-9.195	-7.505
	(-11.727, 7.544)	(-11.878, 7.842)	(-26.764, 8.691)	(-27.086, 12.471)
≥75 yrs	3.184	1.427	5.059	6.458
	(-2.159, 8.555)	(-4.053, 6.936)	(-5.112, 15.334)	(-4.852, 17.897)

Appendix C.3 Estimates of percentage changes (95% CI) in ED visits derived from the standard approach, presented by disease categories, age groups, the single- or two-pollutant models, and seasons per 10 $\mu g/m^3$ increase for $PM_{2.5}$

	Effect of $PM_{2.5}$ on ED visits					
	All-seasons		Summer		Winter	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
Cardiovascular						
All-ages	0.144	0.272	3.585	3.232	0.315	1.208
	(-1.109, 1.398)	(-1.007, 1.554)	(-1.014, 8.206)	(-1.831, 8.322)	(-1.954, 2.590)	(-2.242, 4.669)
<65 yrs	-0.823	-0.664	3.606	4.007	-0.034	1.538
	(-2.736, 1.093)	(-2.613, 1.290)	(-3.244, 10.504)	(-3.536, 11.607)	(-3.533, 3.477)	(-3.788, 6.892)
≥65 yrs	0.885	0.977	3.503	2.526	0.534	0.920
	(-0.772, 2.545)	(-0.718, 2.675)	(-2.694, 9.739)	(-4.295, 9.394)	(-2.446, 3.522)	(-3.602, 5.463)
Respiratory						
All-ages	-0.146	0.019	3.938	-0.330	1.599	2.039
	(-1.056, 0.763)	(-0.921, 0.958)	(-0.096, 7.988)	(-4.769, 4.128)	(0.042, 3.159)	(-0.318, 4.401)
0–14 yrs	0.572	0.774	3.107	-1.540	1.968	2.217
	(-0.706, 1.852)	(-0.559, 2.109)	(-3.376, 9.633)	(-8.686, 5.657)	(-0.184, 4.125)	(-1.048, 5.492)
15–64 yrs	-1.703	-1.698	2.915	-2.134	0.442	0.989
	(-3.393, -0.010)	(-3.426, 0.033)	(-3.769, 9.644)	(-9.470, 5.256)	(-2.529, 3.422)	(-3.488, 5.486)
≥65 yrs	1.174	1.526	6.787	4.259	2.468	2.773
	(-0.839, 3.191)	(-0.543, 3.600)	(-1.310, 14.949)	(-4.656, 13.255)	(-1.004, 5.953)	(-2.464, 8.038)

Appendix C.4 Estimates of percentage changes (95% CI) in ED visits derived from standard approach, presented by disease categories, age groups, the single- or two-pollutant models, and seasons per 10 ppb increase for O_3

	Effects of O_3 on ED visits			
	All-seasons		Summer	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
Cardiovascular				
All-ages	0.075	0.232	0.770	0.311
	(-0.885, 1.036)	(-0.754, 1.218)	(-0.911, 2.454)	(-1.561, 2.187)
<65 yrs	1.158	1.161	0.152	-0.356
	(-0.298, 2.616)	(-0.333, 2.656)	(-2.377, 2.687)	(-3.173, 2.468)
≥65 yrs	-0.756	-0.474	1.260	0.853
	(-2.032, 0.523)	(-1.785, 0.838)	(-0.989, 3.515)	(-1.653, 3.364)
Respiratory				
All-ages	0.716	0.534	3.505	3.599
	(-0.023, 1.456)	(-0.231, 1.300)	(2.076, 4.935)	(2.004, 5.198)
0–14 yrs	0.930	0.977	3.443	3.727
	(-0.153, 2.014)	(-0.152, 2.107)	(1.239, 5.651)	(1.259, 6.202)
15–64 yrs	0.477	0.078	4.119	4.373
	(-0.832, 1.787)	(-1.266, 1.425)	(1.708, 6.536)	(1.685, 7.068)
≥65 yrs	0.129	-0.053	2.736	2.228
	(-1.468, 1.729)	(-1.702, 1.599)	(-0.261, 5.742)	(-1.116, 5.583)

Appendix C.5 Comparison of estimates of percentage changes (95% CI) in respiratory ED visits derived from four approaches (standard, SLA, nearest and blending), presented by age groups, the single- or two-pollutant models, and seasons per 10 $\mu g/m^3$ increase for $PM_{2.5}$

	Effect of $PM_{2.5}$ on respiratory ED visits							
	Standard		SLA		Nearest		Blending	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
Summer								
All-ages	3.938	-0.330	3.97	-0.333	3.577	0.514	4.503	0.373
	(-0.096, 7.988)	(-4.769, 4.128)	(-0.098, 7.987)	(-4.772, 4.126)	(-0.350, 7.520)	(-3.843, 4.890)	(0.707, 8.314)	(-3.648, 4.409)
0–14 yrs	3.107	-1.540	3.111	-1.538	4.625	0.905	4.341	0.091
	(-3.376, 9.633)	(-8.686, 5.657)	(-3.373, 9.637)	(-8.684, 5.660)	(-1.594, 10.883)	(-6.035, 7.894)	(-1.715, 10.433)	(-6.314, 6.537)
15–64 yrs	2.915	-2.134	2.910	-2.14	2.588	-0.453	1.581	-2.584
	(-3.769, 9.644)	(-9.470, 5.256)	(-3.775, 9.64)	(-9.476, 5.251)	(-3.949, 9.167)	(-7.680, 6.826)	(-4.718, 7.920)	(-9.255, 4.131)
≥65 yrs	6.787	4.259	6.784	4.255	2.712	0.585	8.736	4.738
	(-1.310, 14.949)	(-4.656, 13.255)	(-1.312, 14.946)	(-4.661, 13.251)	(-5.274, 10.762)	(-8.221, 9.470)	(1.068, 16.463)	(-3.404, 12.947)
Winter								
All-ages	1.599	2.039	1.598	2.038	1.428	1.036	1.796	1.760
	(0.042, 3.159)	(-0.318, 4.401)	(0.041, 3.158)	(-0.319, 4.400)	(-0.095, 2.953)	(-1.015, 3.092)	(0.248, 3.346)	(-0.379, 3.902)
0–14 yrs	1.968	2.217	1.966	2.213	2.548	2.013	3.221	3.518
	(-0.184, 4.125)	(-1.048, 5.492)	(-0.186, 4.123)	(-1.052, 5.489)	(0.444, 4.656)	(-0.830, 4.864)	(1.066, 5.382)	(0.562, 6.482)
15–64 yrs	0.442	0.989	0.442	0.992	-0.509	-2.531	-0.291	-2.434
	(-2.529, 3.422)	(-3.488, 5.486)	(-2.529, 3.423	(-3.485, 5.490)	(-3.428, 2.419)	(-6.438, 1.391)	(-3.240, 2.667)	(-6.498, 1.647)
≥65 yrs	2.468	2.773	2.467	2.772	1.214	3.781	1.219	2.990
	(-1.004, 5.953)	(-2.464, 8.038)	(-1.006, 5.951)	(-2.456, 8.038)	(-2.159, 4.599)	(-0.760, 8.343)	(-2.175, 4.625)	(-1.792, 7.795)

Appendix C.6 Comparison of estimates of percentage changes (95% CI) in respiratory ED visits derived from four approaches (standard, SLA, nearest and blending), presented by age groups and the single- or two-pollutant models per 10 ppb increase for O_3 in the summer

	Effect of O_3 on respiratory ED visits in the summer							
	Standard		SLA		Nearest		Blending	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
All-ages	3.505 (2.076, 4.935)	3.599 (2.004, 5.198)	3.505 (2.077, 4.936)	3.600 (2.005, 5.199)	3.101 (1.747, 4.456)	3.300 (1.783, 4.818)	4.187 (2.885, 5.492)	4.219 (2.819, 5.622)
0–14 yrs	3.443 (1.239, 5.651)	3.727 (1.259, 6.202)	3.444 (1.240, 5.652)	3.728 (1.260, 6.203)	3.442 (1.343, 5.546)	3.403 (1.038, 5.774)	4.110 (2.100, 6.124)	4.199 (2.040, 6.363)
15–64 yrs	4.119 (1.708, 6.536)	4.373 (1.685, 7.068)	4.119 (1.708, 6.536)	4.374 (1.686, 7.069)	3.131 (0.859, 5.409)	3.326 (0.792, 5.867)	4.168 (1.979, 6.362)	4.341 (1.987, 6.701)
≥65 yrs	2.736 (-0.261, 5.742)	2.228 (-1.116, 5.583)	2.736 (-0.261, 5.742)	2.229 (-1.115, 5.584)	2.180 (-0.657, 5.025)	2.801 (-0.363, 5.974)	4.445 (1.699, 7.198)	4.181 (1.220, 7.150)

Appendix C.7 Estimates of percentage changes (95% CI) in daily mortality derived from the blending approach, presented by disease categories, age groups, the single- or two-pollutant models, and seasons per 10 $\mu g/m^3$ increase for $PM_{2.5}$

	Effect of $PM_{2.5}$ on mortality					
	All-seasons		Summer		Winter	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
All-cause						
All-ages	1.057 (-0.619, 2.737)	0.440 (-1.337, 2.220)	0.415 (-5.726, 6.595)	1.738 (-4.822, 8.341)	0.960 (-1.977, 3.906)	1.746 (-2.487, 5.998)
<65 yrs	-0.312 (-4.468, 3.861)	-1.505 (-5.888, 2.897)	-2.274 (-16.669, 12.332)	2.567 (-12.826, 18.201)	-0.518 (-7.887, 6.905)	5.186 (-5.331, 15.815)
65–74 yrs	2.103 (-2.011, 6.233)	1.872 (-2.476, 6.239)	–	–	7.453 (0.111, 14.848)	7.596 (-2.843, 18.145)
≥75 yrs	0.952 (-1.094, 3.002)	0.397 (-1.776, 2.574)	1.865 (-5.733, 9.520)	1.695 (-6.423, 9.879)	-0.246 (-3.803, 3.325)	-0.506 (-5.658, 4.673)
Cardiovascular						
All-ages	1.000 (-1.744, 3.752)	1.172 (-1.745, 4.097)	3.957 (-6.457, 14.479)	2.573 (-8.517, 13.788)	-1.736 (-6.458, 3.008)	2.413 (-4.410, 9.283)
<65 yrs	-4.866 (-14.049, 4.402)	-4.172 (-13.815, 5.565)	6.558 (-24.927, 39.061)	6.849 (-26.578, 41.424)	–	–
65–74 yrs	3.099 (-4.554, 10.812)	2.353 (-5.812, 10.585)	–	–	–	–
≥75 yrs	–	–	2.563 (-9.388, 14.659)	0.687 (-12.045, 13.583)	–	–
Respiratory					–	–
All-ages	-0.317 (-6.034, 5.433)	-1.907 (-8.005, 4.228)	–	–		
<75 yrs	5.059 (-7.037, 17.302)	4.798 (-8.088, 17.852)	–	–	–	–
≥75 yrs	-1.878 (-8.362, 4.649)	-3.844 (-10.762, 3.122)	–	–	–	–

Note: – relative risk could not be estimated as the model was not converged.

Appendix C.8 Estimates of percentage changes (95% CI) in daily mortality derived from the blending approach, presented by disease categories, age groups, the single- or two-pollutant models, and seasons per 10 ppb increase for O_3

	Effect of O_3 on mortality			
	All-seasons		Summer	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
All-cause				
All-ages	-0.476 (-1.665, 0.714)	-0.531 (-1.743, 0.681)	-1.090 (-3.341, 1.166)	-1.406 (-3.834, 1.029)
<65 yrs	-0.917 (-3.785, 1.960)	-1.086 (-4.006, 1.843)	-4.578 (-9.925, 0.797)	-5.093 (-10.852, 0.699)
65–74 yrs	-2.262 (-5.107, 0.590)	-1.844 (-4.739, 1.061)	–	–
≥75 yrs	0.177 (-1.293, 1.649)	0.020 (-1.479, 1.520)	0.367 (-2.434, 3.177)	0.161 (-2.867, 3.198)
Cardiovascular				
All-ages	0.809 (-1.157, 2.780)	0.721 (-1.284, 2.731)	1.253 (-2.511, 5.031)	1.383 (-2.674, 5.457)
<65 yrs	3.769 (-2.588, 10.168)	4.131 (-2.360, 10.664)	-0.573 (-12.442, 11.439)	-0.187 (-12.933, 12.724)
65–74 yrs	-3.383 (-8.747, 2.010)	-3.407 (-8.869, 2.086)	–	–
≥75 yrs	–	–	1.579 (-2.729, 5.905)	1.928 (-2.721, 6.599)
Respiratory				
All-ages	0.857 (-3.247, 4.977)	-0.161 (-4.354, 4.051)	–	–
<75 yrs	0.627 (-7.813, 9.139)	0.418 (-8.174, 9.083)	–	–
≥75 yrs	0.952 (-3.740, 5.666)	-0.319 (-5.120, 4.505)	–	–

Note: –, the relative risk could not be estimated as the model was not converged.

Appendix C.9 Estimates of percentage changes (95% CI) in ED visits derived from the blending approach, presented by disease categories, age groups, the single- or two-pollutant models, and seasons per 10 $\mu g/m^3$ increase for $PM_{2.5}$

	Effect of $PM_{2.5}$ on ED visits					
	All-seasons		Summer		Winter	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
Cardiovascular						
All-ages	1.069	0.869	1.196	1.252	1.706	3.727
	(-0.154, 2.293)	(-0.308, 2.247)	(-3.171, 5.582)	(-3.389, 5.915)	(-0.534, 3.951)	(0.575, 6.889)
<65 yrs	1.151	0.462	0.703	1.172	2.576	3.068
	(-0.721, 3.027)	(-1.487, 2.414)	(-5.809, 7.258)	(-5.745, 8.137)	(-0.913, 6.077)	(-1.795, 7.955)
≥65 yrs	0.862	1.190	1.583	1.277	1.020	4.105
	(-0.753, 2.480)	(-0.501, 2.883)	(-4.298, 7.498)	(-5.025, 7.518)	(-1.902, 3.950)	(-0.33, 7.428)
Respiratory						
All-ages	-0.473	-0.496	4.503	0.373	1.796	1.760
	(-1.367, 0.422)	(-1.435, 0.445)	(0.707, 8.314)	(-3.648, 4.409)	(0.248, 3.346)	(-0.379, 3.902)
0–14 yrs	0.537	0.682	4.341	0.091	3.221	3.518
	(-0.727, 1.802)	(-0.649, 2.016)	(-1.715, 10.433)	(-6.314, 6.537)	(1.066, 5.382)	(0.562, 6.482)
15–64 yrs	-1.845	-2.230	1.581	-2.584	-0.291	-2.434
	(-3.505, -0.182)	(-3.965, -0.491)	(-4.718, 7.920)	(-9.255, 4.131)	(-3.240, 2.667)	(-6.498, 1.647)
≥65 yrs	1.333	1.221	8.736	4.738	1.219	2.990
	(-0.627, 3.297)	(-0.844, 3.290)	(1.068, 16.463)	(-3.404, 12.947)	(-2.175, 4.625)	(-1.792, 7.795)

Appendix C.10 Estimates of percentage changes (95% CI) in ED visits derived from the blending approach, presented by disease categories, age groups, the single- or two-pollutant models, and seasons per 10 ppb increase for O_3

	Effects of O_3 on ED visits			
	All-seasons		Summer	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
Cardiovascular				
All-ages	-0.427	-0.303	0.127	-0.046
	(-1.261, 0.408)	(-1.156, 0.550)	(-1.416, 1.673)	(-1.707, 1.618)
<65 yrs	0.297	0.241	-0.327	-0.491
	(-0.964, 1.560)	(-1.047, 1.531)	(-2.647, 1.998)	(-2.986, 2.011)
≥65 yrs	-0.978	-0.727	0.550	0.386
	(-2.090, 0.136)	(-1.865, 0.411)	(-1.518, 2.622)	(-1.841, 2.618)
Respiratory				
All-ages	0.709	0.484	4.187	4.219
	(0.069, 1.349)	(-0.174, 1.142)	(2.885, 5.492)	(2.819, 5.622)
0–14 yrs	0.639	0.571	4.110	4.199
	(-0.296, 1.574)	(-0.394, 1.538)	(2.100, 6.124)	(2.040, 6.363)
15–64 yrs	0.868	0.596	4.168	4.341
	(-0.265, 2.001)	(-0.562, 1.756)	(1.979, 6.362)	(1.987, 6.701)
≥65 yrs	0.290	0.095	4.445	4.181
	(-1.100, 1.682)	(-1.332, 1.524)	(1.699, 7.198)	(1.220, 7.150)

Appendix C.11 Estimates of percentage changes (95% CI) in daily mortality derived from the blending approach with the 1df model, presented by disease categories, age groups, the single- or two-pollutant models, and seasons per 10 $\mu g/m^3$ increase for $PM_{2.5}$

	Effect of $PM_{2.5}$ on mortality					
	All-seasons		Summer		Winter	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
All-cause						
All-ages	1.077 (-0.599, 2.755)	0.451 (-1.325, 2.230)	0.455 (-5.673, 6.621)	1.825 (-4.719, 8.411)	0.992 (-1.944, 3.937)	1.978 (-2.242, 6.216)
<65 yrs	-0.325 (-4.479, 3.847)	-1.516 (-5.898, 2.885)	-2.752 (-17.103, 11.808)	1.907 (-13.432, 17.484)	-0.523 (-7.895, 6.903)	5.753 (-4.748, 16.364)
65–74 yrs	2.125 (-1.986, 6.253)	1.882 (-2.463, 6.246)	-1.210 (-16.243, 14.071)	1.982 (-14.007, 18.230)	7.391 (0.057, 14.778)	7.248 (-3.144, 17.748)
≥75 yrs	0.972 (-1.073, 3.201)	0.408 (-1.764, 2.584)	1.707 (-5.870, 9.342)	1.597 (-6.498, 9.759)	-0.192 (-3.748, 3.377)	-0.226 (-5.362, 4.938)
Cardiovascular						
All-ages	0.995 (-1.748, 3.746)	1.155 (-1.761, 4.079)	4.168 (-6.230, 14.674)	3.076 (-7.995, 14.270)	-1.777 (-6.498, 2.967)	2.506 (-4.298, 9.356)
<65 yrs	-5.004 (-14.188, 4.265)	-4.361 (-14.006, 5.377)	4.868 (-26.470, 37.215)	5.823 (-27.432, 40.214)	-9.029 (-25.282, 7.495)	-6.022 (-28.542, 17.020)
65–74 yrs	3.177 (-4.471, 10.883)	2.397 (-5.759, 10.621)	11.034 (-17.694, 40.603)	10.710 (-19.779, 42.146)	12.313 (-1.280, 26.091)	17.292 (-2.158, 37.120)
≥75 yrs	1.257 (-1.843, 4.367)	1.528 (-1.770, 4.836)	2.612 (-9.318, 14.686)	1.052 (-11.657, 13.924)	-3.182 (-8.475, 2.139)	1.145 (-6.519, 8.868)
Respiratory						
All-ages	-0.451 (-6.168, 5.299)	-2.070 (-8.167, 4.065)	-1.412 (-23.323, 20.992)	0.039 (-23.364, 24.002)	-2.291 (-11.992, 7.505)	-9.137 (-22.972, 4.894)
<75 yrs	4.792 (-7.314, 17.046)	4.512 (-8.387, 17.579)	-1.401 (-48.704, 48.254)	5.737 (-44.809, 58.959)	-6.232 (-26.999, 14.979)	5.077 (-24.456, 35.504)
≥75 yrs	-1.964 (-8.446, 4.560)	-3.965 (-10.879, 2.997)	-1.311 (-26.082, 24.091)	-1.898 (-28.334, 25.257)	-1.151 (-12.113, 9.933)	-13.527 (-29.145, 2.343)

Appendix C.12 Estimates of percentage changes (95% CI) in daily mortality derived from the blending approach with the 1df model, presented by disease categories, age groups, the single- or two-pollutant models, and seasons per 10 ppb increase for O_3

	Effect of O_3 on mortality			
	All-seasons		Summer	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
All-cause				
All-ages	-0.516	-0.576	-1.114	-1.457
	(-1.704, 0.672)	(-1.786, 0.636)	(-3.356, 1.133)	(-3.874, 0.966)
<65 yrs	-1.012	-1.189	-4.503	-4.902
	(-3.877, 1.861)	(-4.106, 1.737)	(-9.823, 0.845)	(-10.629, 0.858)
65–74 yrs	-2.281	-1.859	-2.839	-3.449
	(-5.122, 0.569)	(-4.752, 1.041)	(-8.173, 2.524)	(-9.163, 2.298)
≥75 yrs	0.147	-0.016	0.321	0.097
	(-1.321, 1.617)	(-1.513, 1.483)	(-2.469, 3.118)	(-2.916, 3.119)
Cardiovascular				
All-ages	0.800	0.699	1.078	1.077
	(-1.165, 2.769)	(-1.305, 2.706)	(-2.670, 4.840)	(-2.961, 5.131)
<65 yrs	3.605	3.929	-1.373	-0.948
	(-2.747, 9.997)	(-2.556, 10.456)	(-13.203, 10.599)	(-13.638, 11.905)
65–74 yrs	-3.423	-3.446	0.881	-0.364
	(-8.782, 1.964)	(-8.902, 2.040)	(-9.289, 11.155)	(-11.249, 10.642)
≥75 yrs	1.203	1.044	1.372	1.593
	(-1.036, 3.446)	(-1.239, 3.331)	(-2.916, 5.679)	(-3.032, 6.240)
Respiratory				
All-ages	0.857	-0.174	-1.656	-1.457
	(-3.242, 4.973)	(-4.363, 4.033)	(-9.610, 6.362)	(-10.027, 7.187)
<75 yrs	0.504	0.252	-7.938	-6.801
	(-7.929, 9.009)	(-8.333, 8.910)	(-23.884, 8.268)	(-23.958, 10.659)
≥75 yrs	0.981	-0.291	0.758	0.654
	(-3.706, 5.690)	(-5.086, 4.527)	(-8.436, 10.038)	(-9.252, 10.659)

Appendix C.13 Estimates of percentage changes (95% CI) in ED visits derived from the blending approach with the 1df model, presented by disease categories, age groups, the single- or two-pollutant models, and seasons per 10 $\mu g/m^3$ increase for $PM_{2.5}$

	Effect of $PM_{2.5}$ on ED visits					
	All-seasons		Summer		Winter	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
Cardiovascular						
All-ages	0.995	0.936	0.898	0.695	1.666	3.606
	(-2.226, 2.219)	(-0.340, 2.213)	(-3.461, 5.276)	(-3.933, 5.346)	(-0.572, 3.909)	(0.466, 6.757)
<65 yrs	1.032	0.384	0.274	0.445	2.556	3.251
	(-0.839, 2.907)	(-1.563, 2.334)	(-6.225, 6.815)	(-6.450, 7.388)	(-0.930, 6.055)	(-1.597, 8.122)
≥65 yrs	0.822	1.188	1.371	0.797	0.953	3.745
	(-0.792, 2.438)	(-0.502, 2.880)	(-4.501, 7.278)	(-5.440, 7.074)	(-1.965, 3.880)	(-0.376, 7.883)
Respiratory						
All-ages	-0.595	-0.562	4.281	-0.180	1.676	1.739
	(-14.88, 0.300)	(-1.500, 0.378)	(0.491, 8.086)	(-4.191, 3.847)	(0.129, 3.225)	(-0.392, 3.874)
0–14 yrs	0.444	0.657	4.328	-0.330	3.068	3.589
	(-0.819, 1.708)	(-0.674, 1.989)	(-1.722, 10.415)	(-6.722, 6.105)	(0.914, 5.227)	(0.643, 6.544)
15–64 yrs	-2.044	-2.384	1.124	-3.512	-0.500	-2.619
	(-3.703, -0.383)	(-4.117, -0.647)	(-5.157, 7.446)	(-10.158, 3.179)	(-3.447, 2.455)	(-6.670, 1.449)
≥65 yrs	1.273	1.200	8.668	4.705	1.306	2.925
	(-0.685, 3.235)	(-0.863, 3.266)	(1.000, 16.395)	(-3.432, 12.909)	(-2.085, 4.708)	(-1.838, 7.710)

Appendix C.14 Estimates of percentage changes (95% CI) in ED visits derived from the blending approach with the 1df model, presented by disease categories, age groups, the single- or two-pollutant models, and seasons per 10 ppb increase for O_3

	Effects of O_3 on ED visits			
	All-seasons		Summer	
	Single-pollutant	Two-pollutant	Single-pollutant	Two-pollutant
Cardiovascular				
All-ages	-2.019	-0.105	0.337	0.228
	(-1.052, 0.615)	(-0.957, 0.747)	(-1.203, 1.879)	(-1.429, 1.887)
<65 yrs	0.576	0.503	-0.092	-0.174
	(-0.683, 1.838)	(-0.783	(-2.406, 2.228)	(-2.662, 2.321)
≥65 yrs	-0.821	-0.575	0.730	0.617
	(-1.933, 0.291)	(-1.711, 0.563)	(-1.333, 2.797)	(-1.604, 2.842)
Respiratory				
All-ages	1.001	0.758	4.488	4.578
	(0.362, 1.641)	(0.101, 1.416)	(3.189, 5.789)	(3.182, 5.977)
0–14 yrs	0.936	0.855	4.483	4.621
	(0.002, 1.870)	(-0.109, 1.821)	(2.480, 6.491)	(2.470, 6.776)
15–64 yrs	1.247	0.950	4.592	4.865
	(0.116, 2.380)	(-0.207, 2.109)	(2.406, 6.781)	(2.518, 7.219)
≥65 yrs	0.445	0.239	4.423	4.151
	(-0.943, 1.835)	(-1.186, 1.665)	(1.686, 7.168)	(1.201, 7.110)

Appendix C.15 Comparison of estimates of percentage changes (95% CI) in daily mortality associated with $PM_{2.5}$ and O_3 exposure between without and with ambient pollution readings on bushfire and dust storm days derived from the standard approach in the all-ages group in the single-pollutant model, presented by disease categories and seasons..

	All-seasons		Summer	
	With	Without	With	Without
$PM_{2.5}$				
All-cause	1.005	-0.082	2.355	-0.175
	(-0.049, 2.059)	(-1.810, 1.649)	(0.587, 4.126)	(-6.604, 6.296)
Cardiovascular	2.422	0.001	3.380	1.317
	(0.685, 4.162)	(-2.823, 2.833)	(0.443, 6.327)	(-9.579, 12.334)
Respiratory	-0.360	-1.441	0.566	-5.567
	(-4.187, 3.482)	(-7.332, 4.486)	(-5.946, 7.120)	(-28.682, 18.098)
O_3				
All-cause	-0.231	-0.595	0.081	-1.371
	(-1.488, 1.028)	(-1.942, 0.755)	(-2.033, 2.199)	(-3.817, 1.080)
Cardiovascular	2.103	1.483	1.895	1.025
	(0.019, 4.191)	(-0.745, 3.716)	(-1.643, 5.446)	(-3.064, 5.131)
Respiratory	2.023	1.958	3.284	1.303
	(-2.364, 6.429)	(-2.713, 6.650)	(-4.349, 10.975)	(-7.483, 10.167)

Note: The estimates are presented per 10 $\mu g/m^3$ increase for $PM_{2.5}$ and per 10 ppb increase for O_3

Appendix C.16 Comparison of estimates of percentage changes (95% CI) in daily ED visits associated with $PM_{2.5}$ and O_3 exposure between without and with ambient pollution readings on bushfire and dust storm days derived from the standard approach in the all-ages group in the single-pollutant model, presented by disease categories and seasons.

	All-seasons		Summer	
	With	Without	With	Without
$PM_{2.5}$				
Cardiovascular	0.475	0.144	1.462	3.585
	(-0.260, 1.212)	(-1.109, 1.398)	(0.203, 2.722)	(-1.014, 8.206)
Respiratory	-0.693	-0.146	0.247	3.938
	(-1.279, -0.107)	(-1.056, 0.763)	(-0.828, 1.323)	(-0.096, 7.988)
O_3				
Cardiovascular	0.397	0.075	1.284	0.770
	(-0.498, 1.293)	(-0.885, 1.036)	(-0.188, 2.758)	(-0.911, 2.454)
Respiratory	0.317	0.716	2.569	3.505
	(-0.38, 1.014)	(-0.023, 1.456)	(1.312, 3.827)	(2.076, 4.935)

Note: The estimates are presented per 10 $\mu g/m^3$ increase for $PM_{2.5}$ and per 10 ppb increase for O_3.

Appendices D–Appendices in Chapter 6

Appendix D.1 P-values derived from the Wald/Z-tests to examine the equality of coefficients among the different age groups

Pollutant	Health endpoint	Disease category	Age group	p-value of Wald/Z-test	
				Summer	Winter
$PM_{2.5}$	Mortality	All-cause	0-64	0.84	0.17
$PM_{2.5}$	Mortality	All-cause	65–74		
$PM_{2.5}$	Mortality	All-cause	≥75		
$PM_{2.5}$	Mortality	CVD	0-64	0.87	0.08
$PM_{2.5}$	Mortality	CVD	65–74		
$PM_{2.5}$	Mortality	CVD	≥75		
$PM_{2.5}$	Mortality	RD	0-74	0.50	0.34
$PM_{2.5}$	Mortality	RD	≥75		
$PM_{2.5}$	ED visits	CVD	0-64	0.40	0.25
$PM_{2.5}$	ED visits	CVD	≥65		
$PM_{2.5}$	ED visits	RD	0–14	0.33	0.15
$PM_{2.5}$	ED visits	RD	15–64		
$PM_{2.5}$	ED visits	RD	≥65		
O_3	Mortality	All-cause	0-64	0.23	NA*
O_3	Mortality	All-cause	65–74		
O_3	Mortality	All-cause	≥75		
O_3	Mortality	CVD	0-64	0.92	NA*
O_3	Mortality	CVD	65–74		
O_3	Mortality	CVD	≥75		
O_3	Mortality	RD	0-74	0.18	NA*
O_3	Mortality	RD	≥75		
O_3	ED visits	CVD	0-64	0.30	NA*
O_3	ED visits	CVD	≥65		
O_3	ED visits	RD	0–14	1.00	NA*
O_3	ED visits	RD	15–64		
O_3	ED visits	RD	≥65		

Note: *CVD* cardiovascular disease, *RD* respiratory disease, *NA* Not applicable

Appendix D.2 Estimates of percentage changes and 95% CI in daily mortality and ED visits derived from the basic and stratification models, presented by disease category, health endpoint, pollutant and season.

Pollutant	Health endpoint	Disease category	Temp cut-point in percentile (in°C)	Estimates and 95% CI from the basic model	Estimates and 95% CI from the stratification model			p-value of Wald test*
					Low stratum	Middle stratum	High stratum	
Summer								
$PM_{2.5}$	Mortality	CVD	15, 70 (15.9, 21.5)	4.168 (-6.230, 14.674)	-8.339 (-30.506, 14.335)	0.096 (-12.962, 13.326)	6.110 (-4.807, 17.146)	0.40
$PM_{2.5}$	Mortality	RD		-1.412 (-23.323, 20.992)	-3.431 (-48.616, 43.899)	-9.898 (-37.420, 18.411)	2.035 (-20.995, 25.607)	0.07
$PM_{2.5}$	ED visits	CVD		0.898 (-3.461, 5.276)	-7.876 (-16.634, 0.960)	-0.964 (-6.187, 4.286)	2.239 (-2.397, 6.895)	0.60
$PM_{2.5}$	ED visits	RD		4.281 (0.491, 8.086)	-4.335 (-11.640, 3.024)	4.304 (-0.182, 8.811)	4.764 (0.715, 8.829)	0.02
O_3	Mortality	CVD		1.078 (-2.670, 4.840)	-2.772 (-8.424, 2.913)	0.720 (-3.209, 4.664)	2.238 (-1.903, 6.396)	0.18
O_3	Mortality	RD		-1.656 (-9.610, 6.362)	-1.313 (-13.151, 10.667)	-2.976 (-11.312, 5.430)	-0.010 (-8.777, 8.835)	<0.01
O_3	ED visits	CVD		0.337 (-1.203, 1.879)	-2.257 (-4.586, 0.078)	-0.133 (-1.736, 1.473)	1.422 (-0.274, 3.121)	0.58
O_3	ED visits	RD		4.488 (3.189, 5.789)	3.730 (1.792, 5.673)	4.983 (3.630, 6.338)	3.930 (2.502, 5.359)	<0.01
Winter								
$PM_{2.5}$	Mortality	CVD	15, 85 (8.4, 11.9)	-1.777 (-6.498, 2.967)	-1.933 (-7.727, 3.896)	-0.253 (-5.559, 5.080)	-10.688 (-21.890, 0.642)	0.15
$PM_{2.5}$	Mortality	RD		-2.291 (11.992, 7.505)	-4.649 (-16.558, 7.404)	0.763 (-10.092, 11.736)	-10.131 (-33.108, 13.392)	0.06
$PM_{2.5}$	ED visits	CVD		1.666 (-0.572, 3.909)	0.070 (-2.757, 2.904)	30.17 (0.519, 5.521)	0.591 (-4.149, 5.352)	0.41
$PM_{2.5}$	ED visits	RD		1.676 (0.129, 3.225)	3.147 (1.221, 5.076)	0.506 (-1.224, 2.239)	2.009 (-1.246, 5.274)	0.01

Note: * Testing the equality of coefficients at the different temperature strata (df=2). *CVD* cardiovascular disease, *RD* respiratory disease

Appendix D.3 Results of the best identified cut-points based on LR tests in Step 2 across the disease categories, health endpoints, and pollutants with different $PM_{2.5}$ and O_3 lag times and O_3 metrics in the summer

Pollutant	Health endpoint	Disease category	Tested parameter	Cut-points[a,e]	First tier[b]		Second tier[c]		Third tier[d]	
					p-value of LR test	Identified cut-points[e]	p-value of LR test	Identified cut-points[e]	p-value of LR test	Identified cut-points[e]
Cross-matching 1 : $PM_{2.5}$ L01 + O_3 L01 + O_3 max 8-h										
$PM_{2.5}$	Mortality	CVD	L01	70, 95 (21.5, 26.2)	0.92	70, 95	0.19	15, 95	0.50	15, 70
$PM_{2.5}$	Mortality	RD	L01	20, 95 (16.4, 26.2)		(21.5, 26.2)		(15.8, 26.2)		(15.8, 21.4)
$PM_{2.5}$	ED visits	CVD	L01	15, 25 (15.8, 16.8)	1.00	5, 15				
$PM_{2.5}$	ED visits	RD	L01	20, 50 (16.3, 19.1)		(14.4, 15.8)				
O_3	Mortality	CVD	L01, max 8-h	15, 85 (15.9, 24.0)	0.56	60, 85	0.27	60, 70		
O_3	Mortality	RD	L01, max 8-h	60, 90 (20.3, 24.9)		(20.2, 24.0)		(20.2, 21.4)		
O_3	ED visits	CVD	L01, max 8-h	15, 70 (15.8, 21.4)	0.60	15, 70				
O_3	ED visits	RD	L01, max 8-h	60, 75 (20.2, 22.0)		(15.8, 21.4)				
Cross-matching 2: $PM_{2.5}$ L01 + O_3 L02 + O_3 max 1-h										
$PM_{2.5}$	Mortality	CVD	L01	70, 95 (21.5, 26.2)	0.92	70, 95	0.19	15, 95	0.42	15, 70
$PM_{2.5}$	Mortality	RD	L01	20, 95 (16.4, 26.2)		(21.5, 26.2)		(15.8, 26.2)		(15.8, 21.4)
$PM_{2.5}$	ED visits	CVD	L01	15, 25 (15.8, 16.8)	1.00	5, 15				
$PM_{2.5}$	ED visits	RD	L01	20, 50 (16.3, 19.1)		(14.4, 15.8)				
O_3	Mortality	CVD	L02, max 1-h	15, 85 (15.9, 24.0)	0.47	60, 70	0.32	15, 70		
O_3	Mortality	RD	L02, max 1-h	40, 90 (18.2, 24.9)		(20.3, 21.5)		(15.8, 21.4)		
O_3	ED visits	CVD	L02, max 1-h	15, 70 (15.8, 21.4)	0.43	15, 70				
O_3	ED visits	RD	L02, max 1-h	60, 75 (20.2, 22.0)		(15.8, 21.4)				

Note: *CVD* cardiovascular disease, *RD* respiratory disease

[a] Each pair of cut-points presented in each row of this column was identified for each disease category, health endpoint and air pollutant; [b] The first tier tested the same cut-points across the disease categories for each health endpoint and pollutant; [c] The second tier tested the same cut-points across the disease categories and the health endpoints for each pollutant; [d] The third tier tested the same cut-points across the disease categories, health endpoints and pollutants; [e] All the temperature cut-points presented in this table were expressed in percentiles with the unit of °C in the parentheses.

Appendix D.3 Results of the best identified cut-points based on LR tests in Step 2 across the disease categories, health endpoints and pollutants with different $PM_{2.5}$ and O_3 lag times and O_3 metrics in the summer (continued)

Pollutant	Health endpoint	Disease category	Tested parameter	Cut-points[a,e]	First tier[b]		Second tier[c]		Third tier[d]	
					p-value of LR test	Identified cut-points[e]	p-value of LR test	Identified cut-points[e]	p-value of LR test	Identified cut-points[e]
Cross-matching 3: $PM_{2.5}$ L02+ O_3 L02 + O_3 max 1-h										
$PM_{2.5}$	Mortality	CVD	L02	70, 85 (21.5, 24.0)	0.07	40, 50	1.00	15, 95	0.35	15, 70
$PM_{2.5}$	Mortality	RD	L02	35, 95 (17.8, 26.2)		(18.2, 19.2)		(15.8, 26.1)		(15.8, 21.4)
$PM_{2.5}$	ED visits	CVD	L02	70, 80 (21.4, 22.9)	0.56	15, 90				
$PM_{2.5}$	ED visits	RD	L02	15, 50 (15.8, 19.1)		(15.8, 24.8)				
O_3	Mortality	CVD	L02,max 1-h	15, 85 (15.9, 24.0)	0.47	60, 70	0.32	15, 70		
O_3	Mortality	RD	L02,max 1-h	40, 90 (18.2, 24.9)		(20.3, 21.5)		(15.8, 21.4)		
O_3	ED visits	CVD	L02,max 1-h	15, 70 (15.8, 21.4)	0.43	15, 70				
O_3	ED visits	RD	L02,max 1-h	60, 75 (20.2, 22.0)		(15.8, 21.4)				
Cross-matching 4: $PM_{2.5}$ L02 + O_3 L01 + O_3 max 8-h										
$PM_{2.5}$	Mortality	CVD	L02	70, 85 (21.5, 24.0)						
$PM_{2.5}$	Mortality	RD	L02	35, 95 (17.8, 26.2)						
$PM_{2.5}$	ED visits	CVD	L02	70, 80 (21.4, 22.9)	0.56	15, 90				
$PM_{2.5}$	ED visits	RD	L02	15, 50 (15.8, 19.1)		(15.8, 24.8)				
O_3	Mortality	CVD	L01,max8-h	15, 85 (15.9, 24.0)	0.56	60, 85	0.27	60, 70		
O_3	Mortality	RD	L01,max8-h	40, 90 (18.2, 24.9)		(20.3, 24.0)		(20.2, 21.4)		
O_3	ED visits	CVD	L01,max8-h	15, 70 (15.8, 21.4)	0.60	15, 70				
O_3	ED visits	RD	L01,max8-h	60, 75 (20.2, 22.0)		(15.8, 21.4)				

Note: *CVD* cardiovascular disease, *RD* respiratory disease

[a] Each pair of cut-points presented in each row of this column was identified for each disease category, health endpoint and air pollutant; [b] The first tier tested the same cut-points across the disease categories for each health endpoint and pollutant; [c] The second tier tested the same cut-points across the disease categories and the health endpoints for each pollutant; [d] The third tier tested the same cut-points across the disease categories, health endpoints and pollutants; [e] All the temperature cut-points presented in this table were expressed in percentiles with the unit of °C in the parentheses

Appendix D.3 Results of the best identified cut-points based on LR tests in Step 2 across the disease categories, health endpoints and pollutants with different $PM_{2.5}$ and O_3 lag times and O_3 metrics in the summer (continued)

Pollutant	Health endpoint	Disease category	Tested parameter	Cut-points[a,e]	First tier[b]		Second tier[c]		Third tier[d]	
					p-value of LR test	Identified cut-points[e]	p-value of LR test	Identified cut-points[e]	p-value of LR test	Identified cut-points[e]
Cross-matching5: $PM_{2.5}$ L01 + O_3 L02 + O_3 max8-h										
$PM_{2.5}$	Mortality	CVD	L01	70, 95 (21.5, 26.2)	0.92	70, 95 (21.5, 26.2)	0.19	15, 95 (15.8, 26.2)	0.42	15, 70
$PM_{2.5}$	Mortality	RD	L01	20, 95 (16.4, 26.2)						
$PM_{2.5}$	ED visits	CVD	L01	15, 25 (15.8, 16.8)	1.00	5, 15 (14.4, 15.8)				
$PM_{2.5}$	ED visits	RD	L01	20, 50 (16.3, 19.1)						
O_3	Mortality	CVD	L02,max 8-h	30, 85 (17.3, 24.0)	0.04	15, 85 (15.9, 24.0)	0.51	15, 70 (15.8, 21.4)		
O_3	Mortality	RD	L02,max 8-h	40, 90 (18.2, 24.9)						
O_3	ED visits	CVD	L02,max 8-h	15, 70 (15.8, 21.4)	0.28	15, 70 (15.8, 21.4)				
O_3	ED visits	RD	L02,max 8-h	60, 75 (20.2, 22.0)						

Note: *CVD* cardiovascular disease, *RD* respiratory disease

[a] Each pair of cut-points presented in each row of this column was identified for each disease category, health endpoint and air pollutant; [b] The first tier tested the same cut-points across the disease categories for each health endpoint and pollutant; [c] The second tier tested the same cut-points across the disease categories and the health endpoints for each pollutant; [d] The third tier tested the same cut-points across the disease categories, health endpoints and pollutants; [e] All the temperature cut-points presented in this table were expressed in percentiles with the unit of °C in the parentheses.

Appendix D.4 Results of the best identified cut-points based on LR tests in Step 2 across the disease categories, health endpoints, and pollutants with different $PM_{2.5}$ and O_3 lag times and O_3 metrics in the winter

Pollutant	Health endpoint	Disease category	Tested parameter	Cut-points[a,e]	First tier[b]		Second tier[c]		Third tier[d]	
					p-value of LR test	Identified cut-points[e]	p-value of LR test	Identified cut-points[e]	p-value of LR test	Identified cut-points[e]
$PM_{2.5}$	Mortality	CVD	L01	75, 90 (11.2, 12.7)	0.95	75, 85 (11.2, 11.9)	0.09	15, 85 (8.3, 11.9)	NA	NA
$PM_{2.5}$	Mortality	RD	L01	75, 85 (11.2, 11.9)						
$PM_{2.5}$	ED visits	CVD	L01	5, 15 (7.5, 8.3)	1.00	5, 15 (7.5, 8.3)				
$PM_{2.5}$	ED visits	RD	L01	5, 15 (7.5, 8.3)						

Note: *CVD* cardiovascular disease, *RD* respiratory disease

[a] Each pair of cut-points presented in each row of this column was identified for each disease category, health endpoint and air pollutant; [b] The first tier tested the same cut-points across the disease categories for each health endpoint and pollutant; [c] The second tier tested the same cut-points across the disease categories and the health endpoints for each pollutant; [d] The third tier tested the same cut-points across the disease categories, health endpoints and pollutants; [e] All the temperature cut-points presented in this table were expressed in percentiles with the unit of °C in the parentheses..

Appendices E–Appendices in Chapter 8

Appendix E.1 Three emission scenarios (adapted from Appendix A of Walsh et al. 2013)

A. Most likely future (MLF) scenario

Although this scenario has been called the 'most likely future', it is just one scenario out of a vast number that are possible. To ensure the scenario was realistic, the best available data were obtained, and all assumptions were subject to scrutiny by scientific and policy officers.

Information on future transport links, industrial activity and traffic volumes was obtained by contacting state and commonwealth agencies. Information on future population, energy use, transport technology and emission controls was obtained from published reports.

- Industry

The 2009 National Pollutant Inventory was used to identify any new industries not present in 2006. Industry growth rates for each sector were available to 2018; extrapolation was used to obtain 2030 estimates. Changes in the power generation sector are expected, with some major coal-fired power stations likely to close. Co-generation and trigeneration are expected to increase significantly, especially in hospitals, shopping centres, some office buildings and industrial sites, recreation centres, universities and new housing estates.

- Transport

Significant changes are expected in the vehicle fleet, fuels used and the level of transport activity (both freight and passenger). The fleet turnover rate was assumed to remain constant. Diesel and petrol will still be widely used, along with less common fuels (natural gas and biofuels). An increasing number of hybrid and electric vehicles are expected, although these will remain a minor component of the fleet. It is assumed that ethanol blends will only be used in a small fraction of petrol vehicles in 2030.

The emission of pollutants from individual vehicles is expected to reduce through new standards (Euro 5 and 6 for light vehicles, and Euro V for heavy vehicles). Overall, the effect of the new standards is expected to outweigh the growth in travel, so total vehicle

exhaust emissions should reduce by 2030. However, road dust emissions are expected to grow in line with traffic volumes.

Shipping, railway and aircraft activity are expected to significantly increase, driven by population growth and consumption patterns. In 2030, Melbourne airport is predicted to become the largest source of formaldehyde in Victoria, as road transport VOC emissions will have significantly reduced.

- Domestic/commercial

Technological innovation to control air emissions in the domestic and small business sector tends to be slow. Therefore emissions from most domestic and commercial sectors (fuel consumption, surface coatings *etc.*) will be directly affected by increased population. Corresponding growth in housing units is expected to drive increases in emissions from domestic lawn mowing and waste burning.

Domestic wood consumption (in heaters and stoves) is a significant source of pollutants in winter in Victoria. Although the number of housing units is increasing, ABS fuel use projections to 2030 indicate a decrease in per-capita consumption of wood, as people switch to electricity and natural gas for heating. The net effect is little change from 2006 to 2030 in total wood combustion.

Although emissions from the domestic/commercial sector have been compiled using published emission factors and best available estimates of activity, significant uncertainty remains in the estimates for this sector.

B. Low impact future scenario

The Green scenario was derived from MLF by making a number of plausible reductions in emissions. Note that these are hypothetical initiatives and do not represent any state or national policy positions.

- In-service wood heaters compliant with AS/NZS 4013 (PM_{10} emissions less than 4g/(kg of dry fuel)).
- All 2-stroke lawn mowers phased out and replaced with 4-stroke mowers.
- 50% of passenger vehicles replaced with electric vehicles.

- 30% less exhaust and evaporative emissions through better maintenance of light vehicles.
- Briquettes phased out and replaced with natural gas of equivalent energy content.
- 90% reduction in SO_2 emissions from Anglesea power station.

C. *High impact future scenario*

Similarly the Brown scenario was derived from MLF through plausible increases in emissions, with the addition of a change to population distribution:

- All trucks running on diesel; no hybrids, no CNG.
- Euro 5 and Euro 6 switched off for all light vehicles.
- Additional co-generation within the Port Phillip Region.
- Increased shipping freight at the Port of Hastings and Port of Melbourne.
- 25% more wood burnt in wood heaters.
- 50% of outer Melbourne population growth redistributed to inner Melbourne suburbs.

Appendix E.2 Correlation matrix of the log relative risks presented in Table 8.2, presented by air pollutant and season

(a) $PM_{2.5}$ in summer

Low temperature stratum	1.00		
Middle temperature stratum	0.57	1.00	
High temperature stratum	0.32	0.61	1.00

(b) $PM_{2.5}$ in winter

Low temperature stratum	1.00		
Middle temperature stratum	0.75	1.00	
High temperature stratum	0.13	0.35	1.00

(c) O_3 in summer

Low temperature stratum	1.00		
Middle temperature stratum	0.65	1.00	
High temperature stratum	0.15	0.24	1.00

Appendix E.3 Estimated changes (95% CI) induced by climate change between decades 3 and 1 in $PM_{2.5}$ and O_3 concentrations, respiratory ED visits associated with exposure to $PM_{2.5}$ and O_3 per day and per season and percentage change of respiratory ED visits per day predicted by 4 GCMs and presented by season

	CSIRO		ECHAM5		GFDL		UKMO	
	Summer	Winter	Summer	Winter	Summer	Winter	Summer	Winter
$PM_{2.5}$								
Change in concentration ($\mu g/m^3$)	0.28	0.05	0.26	-0.04	0.15	0.26	0.20	-0.11
Change in respiratory ED visits per day	0.29 (0.03, 0.55)	-0.02 (0.00, -0.04)	0.31 (0.03, 0.60)	-0.06 (0.00, -0.11)	0.09 (0.01, 0.18)	0.22 (0.02, 0.43)	0.16 (0.02, 0.33)	-0.15 (-0.01, -0.29)
Change in respiratory ED visits per season*	26 (3, 50)	-2 (-0, -4)	28 (3, 54)	-5.2 (-0, -10)	8.0 (1, 16)	20 (2, 39)	15 (2, 30)	-14 (-1,-27)
% change in respiratory ED visits per day	8.0 (7.9, 8.1)	-1.1 (-1.1, -1.0)	8.9 (8.8, 9.0)	-2.3 (-2.3, -2.3)	2.5 (2.4, 2.6)	10.0 (10.0, 10.0)	4.4 (4.2, 4.6)	-6.6 (-6.6,-6.6)
O_3								
Change in concentration (ppb)	0.84	NA	0.96	NA	1.15	NA	0.67	NA
Change in respiratory ED visits per day	0.79 (0.53, 1.07)	NA	0.68 (0.46, 0.92)	NA	1.22 (0.83, 1.65)	NA	0.89 (0.60, 1.22)	NA
Change in respiratory ED visits per season*	71 (48, 96)	NA	61 (41, 83)	NA	110 (74, 149)	NA	80 (54, 110)	NA
% change in respiratory ED visits per day	3.7 (3.6, 3.8)	NA	3.2 (3.1, 3.3)	NA	5.8 (5.7, 6.0)	NA	4.2 (4.0, 4.4)	NA

Note: *90 days in summer (December to February) and 92 days in winter (June to August) per season

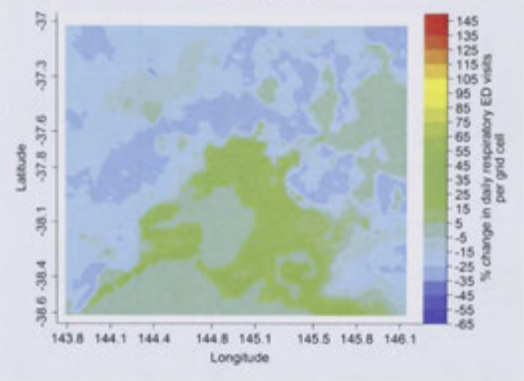
Appendix E.4 Estimated changes (95% CI) induced by climate change taking account of temperature modifying effect between decades 1 and 3 in respiratory ED visits associated with exposure to $PM_{2.5}$ and O_3 per day and per season and percentage change of respiratory ED visits per day predicted by 4 GCMs and presented by season

	CSIRO		ECHAM5		GFDL		UKMO	
	Summer	Winter	Summer	Winter	Summer	Winter	Summer	Winter
$PM_{2.5}$								
Change in respiratory ED visits per day	0.57 (-0.25, 1.37)	0.67 (-1.23, 2.59)	0.28 (-0.14, 0.70)	0.35 (-0.64, 1.37)	0.49 (-0.38, 1.30)	0.68 (-0.94, 2.30)	0.51 (-0.34, 1.36)	0.46 (-1.00, 1.95)
Change in respiratory ED visits per season*	51 (-22, 123)	62 (-113, 238)	25 (-12, 63)	32 (-59, 126)	43 (-34, 117)	62 (-86, 211)	46 (-30, 123)	42 (-92, 179)
% change in respiratory ED visits per day	15.9 (-12.1, 113.9)	46.8 (-906.1, 598.3)	8.2 (-11.5, 36.2)	22.0 (-374.1, 326.7)	14.6 (-24.0, 133.2)	48.7 (-694.0, 528.9)	14.2 (-18.4, 114.4)	31.3 (-620.8, 616.2)
O_3								
Change in respiratory ED visits per day	0.03 (-4.59, 5.34)	NA	-0.09 (-2.23, 2.41)	NA	0.75 (-4.30, 6.45)	NA	0.27 (-4.37, 5.64)	NA
Change in respiratory ED visits per season*	3 (-412.7, 480.3)	NA	-8 (-201.0, 217.0)	NA	68 (-387.3, 580.4)	NA	24 (-393.6, 507.7)	NA
% change in respiratory ED visits per day	0.2 (-36.3, 17.9)	NA	-0.4 (-16.3, 8.7)	NA	3.6 (-32.2, 23.9)	NA	1.3 (-33.3, 19.8)	NA

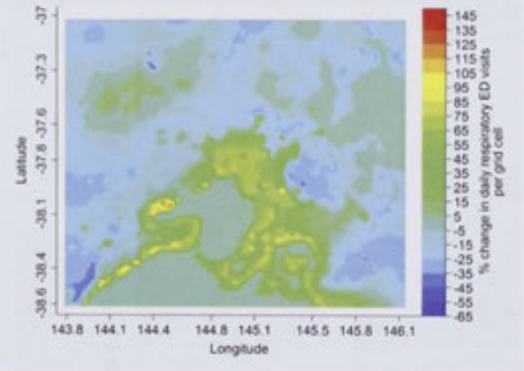
Note: *90 days in summer (December to February) and 92 days in winter (June to August) per season

Appendix E.5 Percentage change in the number of daily respiratory ED visits between decades 1 and 3 with including the temperature modifying effect predicted by the ECHAM5, GFDL and UKMO models

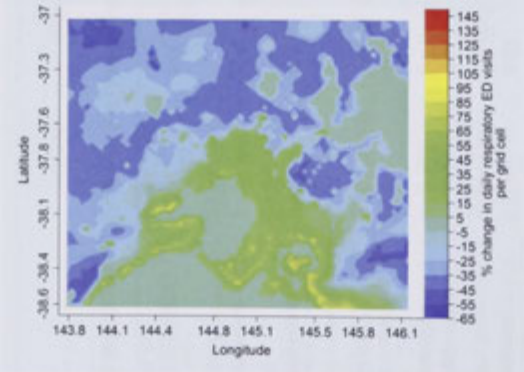
(a) ECHAM5



(b) GFDL



(c) UKMO



Appendix E.6 Estimated changes (95% CI) between decade 1 and 2 in $PM_{2.5}$ and O_3 concentrations, respiratory ED visits associated with exposure to $PM_{2.5}$ and O_3 per day and per season and percentage change of respiratory ED visits per day (unadjusted and adjusted for population) predicted by the CSIRO model under Groups A to C presented by season

	A		B		C1		C2		C3	
	Summer	Winter	Summer	Winter	Summer	Winter	Summer	Winter	Summer	Winter
$PM_{2.5}$										
Change in concentration ($\mu g/m^3$)	0.09	0.20	0.09	0.20	-0.13	-0.07	-0.03	0.10	0.08	0.22
Change in respiratory ED visits per day	0.08 (0.01,0.16)	0.18 (0.01,0.36)	1.30 (0.15,2.48)	0.84 (0.07,1.63)	0.91 (0.10,1.73)	0.37 (0.03,0.72)	1.09 (0.12,2.08)	0.71 (0.06,1.38)	1.50 (0.17,2.87)	1.06 (0.08,2.06)
Change in respiratory ED visits per season*	8 (1,15)	17 (1,33)	117 (13,223)	78 (6,150)	82 (9,156)	34 (3,67)	99 (11,187)	66 (5,127)	135 (15,258)	98 (8,189)
% change in daily respiratory ED vistis**	3.1 (3.1,3.1)	12.8 (12.7,12.9)	48.1 (48.1,48.2)	58.6 (58.5,58.7)	33.7 (33.6,33.9)	26.0 (26.0,26.1)	40.4 (40.3,40.5)	49.5 (49.5,49.6)	55.5 (55.5,55.6)	73.8 (73.6,74.0)
% change in daily respiratory ED visits***	3.1 (3.1,3.1)	12.8 (12.7,12.9)	2.3 (2.3,2.3)	9.6 (9.5,9.7)	-7.6 (-7.7,-7.5)	-12.9 (-12.8,-12.9)	-3.0 (-3.0,-3.0)	3.3 (3.3,3.4)	7.5 (7.4,7.5)	20.1 (19.9,20.2)
O_3										
Change in concentration (ppb)	0.43	NA	0.43	NA	-0.87	NA	-0.51	NA	0.10	NA
Change in respiratory ED visits per day	0.33 (0.22,0.44)	NA	10.15 (7.04,13.37)	NA	12.18 (8.44,16.08)	NA	12.36 (8.56,16.33)	NA	11.53 (7.99,15.22)	NA
Change in respiratory ED visits per season*	30 (20,40)	NA	913 (634,1203)	NA	1096 (759,1447)	NA	1113 (770,1470)	NA	1038 (719,1370)	NA
% change in daily respiratory ED visits**	1.5 (1.5,1.6)	NA	47.6 (47.6,47.7)	NA	57.2 (57.0,57.4)	NA	58.0 (57.8,58.3)	NA	54.1 (54.0,54.3)	NA
% change in daily respiratory ED visits***	1.5 (1.5,1.6)	NA	2.0 (2.0,2.1)	NA	8.6 (8.5,8.8)	NA	9.2 (9.1,9.4)	NA	6.5 (6.4,6.6)	NA

Note:*90 days in summer (December to February) and 92 days in winter (June to August) per season; ** unadjusted population; *** adjusted population;
A–Climate change only; B–Climate change + 2030 MLF population growth; C1–Climate change + 2030 MLF population growth + low impact emissions;
C2–Climate change + 2030 MLF population growth +MLF emissions; C3–Climate change + 2030 MLF population growth +high impact emissions

Appendix E.7 Estimated changes (95% CI) between decades 1 and 2 in respiratory morbidity associated with exposure to $PM_{2.5}$ per day and per season and percentage change of respiratory morbidity per day (unadjusted and adjusted for population) by the CSIRO model under Groups D and E presented by season

	D	E1[a]	E2[b]	E3[c]	E4[d]
$PM_{2.5}$ in summer					
Change in respiratory morbidity[†] per day	1.18 (0.19, 2.11)	1.88 (0.26, 3.49)	1.61 (-0.04 ,3.40)	0.51 (-0.27, 1.38)	3.48 (0.92, 6.49)
Change in respiratory morbidity per season[‡]	106 (17, 190)	169 (23, 315)	145 (-4, 306)	46 (-25, 125)	313 (82, 584)
% change in daily respiratory morbidity[**]	43.9 (29.0, 86.7)	70.0 (12.0, 244.1)	65.1 (-34.8, 269.6)	21.0 (-22.9, 58.1)	57.6 (2.4, 251.5)
% change in daily respiratory morbidity[***]	-0.5 (-10.9, 29.1)	-2.9 (-3.1, -2.9)	-2.9 (-3.1, -2.9)	-3.0 (-3.0, -2.9)	-3.0 (-3.2, -2.9)
$PM_{2.5}$ in winter					
Change in respiratory morbidity[†] per day	0.68 (-0.50, 1.83)	0.54 (-0.37, 1.45)	0.82 (-0.10, 1.83)	1.50 (0.70, 2.31)	1.74 (0.28, 3.22)
Change in respiratory morbidity per season[‡]	62 (-46, 168)	50 (-35, 134)	75 (-9, 169)	138 (65, 212)	160 (26, 296)
% change in daily respiratory morbidity[**]	73.8 (-383.9, 406.3)	32.8 (-42.4, 80.6)	56.8 (-13.6, 210.7)	53.4 (30.6, 101.9)	82.2 (29.9, 134.0)
% change in daily respiratory morbidity[***]	20.1 (-296.2, 250.0)	3.3 (3.3, 3.3)	3.3 (3.3, 3.3)	3.3 (3.3, 3.3)	3.3 (3.3, 3.4)

Note:*90 days in summer (December to February) and 92 days in winter (June to August) per season; ** unadjusted population; *** adjusted population;
[†] Groups D, E1, E2 and all the sub-groups under Group F examined changes in respiratory ED visits whereas Groups E3 and E4 examined changes in respiratory hospital admissions;
D–Climate change + 2030 MLF population growth + MLF emissions + temperature modifying effect;
E–Climate change + 2030 MLF population growth + MLF emissions + age distribution + choice of dose-response functions;
[a] Risk estimates for Group E1 were derived from the blending approach presented in Chapter 5; [b] Risk estimates for Group E2 were derived from the standard approach presented in Chapter 5; [c] Risk estimates for Group E3 were derived from the EPHC study; [d] Risk estimates for Group E4 were derived from a study conducted by EPA Victoria.

Appendix E.8 Estimated changes (95% CI) between decades 1 and 2 in respiratory morbidity associated with exposure to O_3 per day and per season and percentage change of respiratory morbidity per day (unadjusted and adjusted population) predicted by the CSIRO model under Groups D to F presented by season

	D	E1[a]	E2[b]	E3[c]	E4[d]	F1	F2	F3
Change in respiratory morbidity[*] per day	12.03 (4.43, 20.57)	13.57 (8.09, 19.43)	9.52 (3.91, 15.74)	4.29 (-1.28, 10.32)	8.09 (2.89, 13.84)	8.91 (6.21, 11.70)	4.98 (3.21, 6.79)	1.38 (0.54, 2.24)
Change in respiratory morbidity per season[*]	1083 (398, 1851)	1221 (729, 1749)	857 (352, 1417)	387 (-116, 929)	728 (260, 1246)	802 (559, 1053)	448 (289, 611)	124 (49, 201)
% change in daily respiratory morbidity[**]	56.3 (38.3, 65.5)	62.9 (45.7, 82.2)	57.3 (31.7, 80.8)	28.7 (-15.6, 52.8)	49.7 (29.8, 89.0)	64.2 (64.0, 64.4)	80.8 (80.6, 80.9)	115.5 (115.3, 115.6)
% change in daily respiratory morbidity[***]	8.1 (-4.4, 14.4)	9.3 (9.1, 9.5)	9.1 (9.0, 9.4)	9.4 (9.1, 10.0)	9.2 (8.9, 9.7)	13.5 (13.4, 13.7)	24.9 (24.8, 25.0)	48.92 (48.8, 49.0)

Note:*90 days in summer (December to February) per season; ** unadjusted population; *** adjusted population;

[*] Groups D, E1, E2, and all the sub-groups under Group F examined changes in respiratory ED visits whereas Groups E3 and E4 examined changes in respiratory hospital admissions;

D–Climate change + 2030 MLF population growth + MLF emissions + temperature modifying effect;

E–Climate change + 2030 MLF population growth + MLF emissions + age distribution + choice of dose-response functions;

F–Climate change + 2030 population growth + MLF emissions + O3 threshold levels (F1=10 ppb, F2=20 ppb and F3=30 ppb);

[a] Risk estimates for Group E1 were derived from the blending approach presented in Chapter 5; [b] Risk estimates for Group E2 were derived from the standard approach presented in Chapter 5; [c] Risk estimates for Group E3 were derived from the EPHC study; [d] Risk estimates for Group E4 were derived from a study conducted by EPA Victoria.